This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

The

Massachusetts Medical Society

Librarian

MEDICAL COMMUNICATIONS

OF

THE MASSACHUSETTS MEDICAL SOCIETY.

WITH AN APPENDIX,

CONTAINING THE PROCEEDINGS OF THE COUNCILLORS AND OF THE SOCIETY.

VOLUME XX.

IN THREE PARTS.

BOSTON:
PRINTED FOR THE SOCIETY.
PRESS OF DAVID CLAPP & SON.
1907.

CONTENTS.

ART.		PAGE.
I.	Annual Discourse for 1905.—Some Facts and Fancies about Mind and Body. By CHARLES A. DREW, M.D., of Bridgewater	1
II.	The Shattuck Lecture.—Some Problems of Intermediary Metabolism. By RUSSELL H. CHITTENDEN, Ph.D., of New Haven, Conn.	27
III.	What the Suburban Surgeon is Doing in the Abdomen, and How He Does It. By CHARLES E. DURANT, M.D., of Haverhill	71
IV.	Causes of Disability after Fractures of the Lower Leg and Ankle. By FREDERIC J. COTTON, M.D., of Boston	105
V.	Early Diagnosis of Surgical Disease in the Urinary Organs. By BENJAMIN TENNEY, M.D., of Boston	119
VI.	Empyema of the Frontal Sinus. By FREDERIC C. COBB, M.D., of Boston	137
VII.	The Applied Anatomy of the Frontal Sinus. By HARRIS P. MOSHER, M.D., of Boston.	147
VIII.	Some Interesting Features connected with the Outbreak of Small Pox at North Adams, Mass., in 1904. By LYMAN A. JONES, M.D., of North Adams	165
IX.	Ocular Vertigo of Interest to the General Practitioner. By BENJAMIN P. CROFT, M.D., of Greenfield	177
X.	The Medical Treatment of Gastric Ulcer and Hyperchlorhydria. By FREDERICK C. SHATTUCK, M.D., of Boston	189
XI.	A Clinical and Laboratory Study of the Therapeutic Value of Hydrochloric Acid in Diseases of the Stomach. By RICHARD F. CHASE, M.D., of Brookline	203
XII.	Methods in Medicine. By LEWELLYS F. BARKER, M.D., of Chicago, Ill.	225

XIII.—A Series of Demonstrations illustrating Methods of Diagnosis in Medicine and Surgery, and also the Causes and Lesions of Disease. By WILLIAM T. COUNCILMAN, M.D., of Boston 2 53

XIV.—Annual Discourse for 1906.—The General Practitioner and the Specialist. By JOHN L. HILDRETH, M.D., of Cambridge . . . 277

XV.—The Shattuck Lecture.—A Contribution to the Chemistry of the Bacterial Cell and a Study of the Effects of some of the Split Products on Animals. By VICTOR C. VAUGHAN, M.D., of Ann Arbor, Mich. . . . 295

XVI.—Removal of an Enormous Ovarian Tumor. By WALTER A. SMITH, M.D., of Springfield . 349

XVII.—Diseases of the Biliary Passages and Pancreas. By ELBRIDGE G. CUTLER, M.D., of Boston 353

XVIII.—Diseases of the Bile Passages, including the Liver, Gall Bladder and Pancreas. By MAURICE H. RICHARDSON, M.D., of Boston . 363

XIX.—Annual Discourse for 1907.—The Segregation of Consumptives. By J. F. ALLEYNE ADAMS, M.D., of Pittsfield 397

XX.—The Shattuck Lecture.—The Past, Present, and Future of Tuberculosis. By FREDERICK C. SHATTUCK, M.D., of Boston . . 417

XXI.—The Channels of Infection in Tuberculosis, together with Some Remarks on the Outlook concerning a Specific Therapy. By THEOBALD SMITH, M.D., of Boston . . . 447

XXII.—The Organization of Tuberculosis Classes. By JOSEPH H. PRATT, M.D., of Boston . . 473

XXIII.—Medical Inspectors: Their Function. By CHARLES HARRINGTON, M.D., of Boston . 493

XXIV.—Factory Inspection. By FRANK G. WHEATLEY, M.D., of North Abington . . . 501

XXV.—Lead Poisoning in a Rural Community. By WILLIAM N. COWLES, M.D., of Ayer . 511

XXVI.—Stenosis of the Pylorus in Infancy. By CHARLES L. SCUDDER, M.D., of Boston . 525

XXVII.—The Roentgen Ray in Pediatrics. By THOMAS M. ROTCH, M.D., of Boston . . . 541

XXVIII.—Subacute Polymyositis, with a Report of Three Cases. By BENJAMIN T. BURLEY, M.D., of Worcester 553

XXIX.—The Operative Treatment of Varicose Veins of the Lower Extremity. By JOHN T. BOTTOMLEY, M.D., of Boston 575

XXX.—The Treatment of Typhoid Fever with Specific Sera, Filtrates and Residues (Vaughan). By MARK W. RICHARDSON, M.D., of Boston . 593

XXXI.—Experience with Opsonins and Bacterial Vaccines in the Treatment of Tuberculous and Non-Tuberculous Arthritis. By CHARLES F. PAINTER, M.D., of Boston 613

XXXII.—The Prostate Gland from the Surgical Standpoint. By CHARLES S. BENSON, M..D, of Haverhill. 635

XXXIII.—Twenty-five Years' Experience in the Treatment of Stricture of the Urethra. By FRANCIS S. WATSON, M.D., of Boston . . 651

XXXIV.—Bacterial Opsonins and Vaccines. By TIMOTHY LEARY, M.D., and LEON S. MEDALIA, M.D., of Boston 669

ARTICLE I.

THE ANNUAL DISCOURSE.

SOME FACTS AND FANCIES ABOUT MIND AND BODY.

By CHARLES A. DREW, M.D.
OF BRIDGEWATER.

DELIVERED JUNE 14, 1905.

NOTE.—At an Adjourned Meeting of The Massachusetts Medical Society, held Oct. 3, 1860, it was

Resolved, "That The Massachusetts Medical Society hereby declares that it does not consider itself as having endorsed or censured the opinions in former published Annual Discourses, nor will it hold itself responsible for any opinions or sentiments advanced in any future similar discourses."

Resolved, "That the Committee On Publications be directed to print a statement to that effect at the commencement of each Annual Discourse which may hereafter be published."

SOME FACTS AND FANCIES ABOUT MIND AND BODY.

Mr. President and Fellows
of The Massachusetts Medical Society:—

"Electricity is not a form of energy, any more than water is a form of energy. Water may be a vehicle of energy when at a high level or in motion; so may electricity. Electricity cannot be manufactured, as heat can; it can only be moved from place to place, like water; and its energy must be in the form of motion or of strain. Electricity under strain constitutes 'charge'; electricity in locomotion constitutes a current and magnetism; electricity in vibration constitutes light. What electricity itself is we do not know, but it may, perhaps, be a form or aspect of matter. So have taught for thirty years the disciples of Clerk-Maxwell.

Now we may go one step further and say matter is composed of electricity and nothing else."

The quotation I have read you from the pen of Sir Oliver Lodge, as it appeared in Harper's Magazine for August, 1904, must serve me as introduction and text, from which I reserve the right to wander wide, as do some theologians. I would refer you for a complete elucidation of the "Electronic Theory of Matter,"—which I shall only touch in its relation to my theme,—to Sir Oliver Lodge's unabridged thesis, in the "Scientific American Supplements," Nos. 1428 to 1434 inclusive, and to "Modern

Theory of Physical Phenomena," by Augustus Rigbi, as translated by Augustus Trowbridge, and published by the Macmillan Company in 1904.

As my subject deals largely with theories, I may quote you in partial justification that "theory originally fashions science out of facts, and is the indispensable precondition of every important scientific advancement."

Without further introduction or attempt to disarm you, I submit my story.

Some years ago a personal friend, a physician of the best type, and one of the senior members of this Society, talked to me of some day dreams of his early manhood. At a certain age he supposed there must be men living wise enough to explain all the mysteries of life and mind. The phenomena of mind seemed included in those of life, but conscious life, or self-consciousness, seemed an additional problem, and a superadded mystery. He could not then see how these problems could be solved by the laws of molecular physics; and after many years of study, with mind keenly alert and open to the evidence from chemical and physiological laboratories, the mystery to him continues supremely interesting, but as much a mystery as in the days of his early boyhood.

As a boy, he looked for some great preacher or philosopher to explain these mysteries. As he left his rural home, the village preacher and the academy teachers to sit at the feet of college professors, his expectation was keen and his faith ascendant. His acute interest in the problems of nature drew him to physiology and chemistry, while a natural bent and sympathy for humanity led him into the ranks of medicine. In the pride of his knowledge as a college senior there lurked a sense of disappointment, not because he had slighted opportunities, rather because of a dawning suspicion that behind the veil of phenomena were laws and realities the ultimate nature of which he might

never know. As he began his medical studies, smoldering expectation was fanned to a glow, hope rose high again; gross anatomy and the physiology of organs were interesting, but in microscopic anatomy and cell biology, especially as applied to those cells of the central nervous system, lay hidden the veritable elements of the soul. If the riddles of life were not solved, it was held to be due to imperfections of the microscope or to faulty technique; but this was more than forty years ago. The science of physiological psychology was then young and over confident. It pointed with pride to the revelations of its inductive and experimental methods, and, in the assurance of its lusty youth, laughed its older rival, metaphysical psychology, to scorn. Maudsley's thesis on "Body and Mind" had not then been written, but Herbert Spencer's "First Principles" was being widely read, and Charles Darwin's "Origin of Species" had been published several years. The influence of this masterpiece of a master mind was even then shaking the foundations of many cherished beliefs concerning things of earth and heaven. Doubt and apprehension were the first reactions for many conservative minds. Gleesome aggressiveness marked the attitude of others who thought they saw in the theory of evolution the beginning of the end of man's faith and hope in the soul's immortality. "The brain secretes thought and consciousness as the liver secretes bile" and "without phosphorus no thought" were pithy epigrams which served as a rallying cry in the rebellion of the nineteenth century against the older philosophy in favor of a strictly material interpretation of the phenomena of conscious mental life.

It would not be quite true to say that these catch phrases have no potency to-day, but the fallacies they embody have been many times uncovered.

"With phosphorus you light your candle, and with phosphorus you discover Neptune and write the Fifth Symphony;

how charmingly simple and convincing," wrote John Fiske,[1] with thinly veiled irony. "And yet was anything save a bit of rhetoric really gained by singling out phosphorus among the chemical constituents of brain tissue rather than nitrogen or carbon?" "The phosphorus philosophers have often compared thought to a secretion," writes William James.[2] 'The brain secretes thought as the kidneys secrete urine or the liver secretes bile,' are phrases one sometimes hears. The lame analogy need hardly be pointed out. The materials which the brain pours into the blood (cholesterin, creatin, zanthin, or whatever they may be) are the analogues of the bile and urine, being in fact real material excreta. But we know nothing connected with kidney and liver activity which can be in the remotest degree compared with the stream of thought which accompanies the brain's material secretions."

These opinions from the writings of two of the New World's profoundest scholars were written in the dawn of the twentieth century.

In a recent paper on the treatment of paresis, Dr. Edward Cowles[3] writes: "We are learning that inasmuch as psychology can tell us nothing to explain the normal mechanicism of mental activities, we cannot expect to see through the microscope an explanation of diseased thoughts and feelings."

We must not conclude from this that Dr. Cowles was unmindful of the alleged way in which, according to the laws of cerebral association, neural undulations transform themselves into sensations, sensations add themselves together to form feelings, and feelings unite themselves to form ideas, and ideas congregate to form abstract reasoning, etc.; rather we understand the Doctor's meaning to be that physiological psychology and anatomical pathology do not adequately explain. His hope apparently turns to the

[1] Figures refer to notes at end of paper.

deeper underlying plane of physiological chemistry, and he quotes Dr. Otto Folin approvingly as follows: "Microscopically visible structural changes in any tissue or in the cells of any tissue must be preceded by more or less pronounced metabolic changes. Metabolic changes are chemical changes. Chemical changes are the physical exchanges and transmutations that take place in the physical units of matter, the molecules; and the molecules are beyond the ken of the microscopists."

These quotations of a comparatively recent date, from men well informed in that medical specialty most concerned with the phenomena of mind, neither affirm nor deny the material nature of our conscious mental life. They do point to a growing recognition of the limitations of science to unravel mysteries which seem to defy the microscope and, it may be, chemical reagents. They do mark by sharp contrast the modest claims of the older science of to-day from the cock-sure attitude of the youthful science of forty years ago.

It seems,—if we may paraphrase a famous saying of Bacon,—that a superficial knowledge inclines men's minds to make a fetish of science, while a deeper knowledge reveals the limitations and points to the truth that science deals with the causal relation of phenomena, and that physical science takes account only of physical phenomena and their related sequences and is utterly incompetent to pass judgment about things non-material or beyond the limits of man's experience in the body. I have so presumed on the instinctive interest in humanity and its destiny, underneath the scientific finish of members of this Society, as to ask your attention to two modern theories which seem most worthy concerning the relation of the minds and bodies of men.

First, I would call attention to some features of the brain productive or "liver-bile" function theory of the

ultra material psychologists, and, secondly, to some phases of the transmissive brain function theory of Schiller[4] and William James.[5]

I do not know that the liver-bile, brain-thought theory is more commonly held by members of our profession, yet the conclusion easily follows the observation of the coincident development of brain and mind, the lapse of consciousness from pressure on or injury to brain tissue and the greater intelligence of men and animals having the deepest cerebral sulci and the most complex arrangement of cortical gray matter. I doubt if the case of the physiologist has been more strongly put than in the words of Sir Frederick Harrison,[6] who wrote, in defending the so-called "positive philosophy," more than thirty years ago: "Man is one, however compound. Fire his conscience and he blushes. Check his circulation and he thinks wildly or thinks not at all. Impair his secretions and his moral sense is dulled, discolored or depraved; his aspirations flag, his hope, love, faith reel. Impair them still more and he becomes a brute. A few drinks degrades his moral nature to that of a swine. Again, a violent emotion of pity or horror makes him vomit. A lancet will restore him from delirium to clear thought. Excess of thought will waste his sinews. Excess of muscular exercise will deaden thought. An emotion will double the strength of his muscles. And, at last, the prick of a needle or a grain of mineral will in an instant lay to rest forever his body and its unity and all the spontaneous activities of intelligence, feeling and action with which that compound organism was charged."

With a mind tuned to recognize the significance and interdependence of that aggregate of phenomena we call mind and that aggregate of phenomena we call body, we turn eagerly to chemistry, physics and physiology for the justification of our expectations.

This domain of molecular physics populated by hypo-

thetical molecules, divided into hypothetical atoms and subdivided into hypothetical electrons, might well put to shame the fairy dreamland of imaginative childhood.

Chemistry tells us that matter is indestructible. The proof is so conclusive that no scientist has a doubt. As one form of matter is transformed into another, perchance a solid into a liquid or into invisible gases by disturbing molecular relations, so it seems established that the many so-called forces are transmutable one into another. Force being manifested only in motion of matter it would be easy to conclude that no force existed where there was no visible motion. But molecular motion, like some forms of matter, may not be visible and the correlation and equivalence of forces or "the conservation of energy," so-called, represents a doctrine seemingly as impregnable as the axiom of the indestructibility of matter. In searching for a material explanation of the phenomena of life and consciousness we naturally inquire concerning the physical and chemical properties of living matter and matter that has never lived. Again we are told that, so far as the chemist and physicist have knowledge, the distinction is not fundamental, for inorganic bodies and living organisms have very similar chemical elements.

Verworn[7] declares that "the life process consists in the metabolism of proteids," yet carbon, hydrogen, nitrogen, sulphur and oxygen,—elements so conspicuous in inorganic nature,—make up the complex proteid molecule.

While it seems to be established, therefore, that "the vital phenomena of organisms should follow the same general chemical laws as the phenomena of the inorganic world," we should not miss the significance of that supremely important fact that proteids "are never wanting in living substances and are never found in the inorganic world."[8] Hence it is that in the complexity of that proteid molecule seems to lie hidden a key to the mystery of life. And yet,

that molecule is not uniform, we are told by competent chemists. While its elements are but five or six, its atoms vary in position and often number far beyond a thousand,[9]—and the key is not readily found. According to Pfluger,[10] "living proteid does not need to have a constant molecular weight; it is a huge molecule undergoing constant never-ending formation and constant decomposition, and probably behaves toward the usual chemical molecules as the sun behaves toward small meteors."

Then we would further inquire as to the difference, if there be a chemical difference, between the living and the dead proteid molecule. Here, we are told, there is a fundamental difference, as evidenced by a comparison of the decomposition products of living proteid and those obtained by the artificial oxidation of dead proteid. While the non-nitrogenous, or hydro-carbon, decomposition products of the two are essentially the same, the difference in the nitrogenous products of living and dead proteid is marked and important.

It seems, if we may trust Pfluger and Verworn,[11] that in the decomposition products of living proteid may be found that exceedingly poisonous gas cyanogen, which unites with hydrogen to form the deadly hydrocyanic acid and with oxygen and metallic bases to form the highly explosive fulminates. Cyanogen, it seems, is formed by the union of a single atom each of carbon and nitrogen. It acts as a radical in chemical compositions, and is noted among chemists for the intensity of its internal energy.

All chemists may not agree with Verworn and Pfluger[12] that living proteid is distinguished from dead proteid most of all by the vivifying presence of that fiercely energetic radical cyanogen;—yet if it be established beyond peradventure, it is a curious fact that a radical so closely related to the deadly prussic acid and the explosive fulminates should be the essential chemical representative of life.

If we agree with that axiom of physics that every manifestation of force is a mode of motion, molecular or molar, and consent to that other dictum of science that each mode of motion persists until transformed into some other mode of motion, then we are ready to inquire what becomes of the neural currents passing almost continuously from periphery to centre through nerves of special sense. If you hold that these currents are dancing molecules of matter, each unit imparting its motion to the molecule next nearer the centre, as physicists have taught,—or agree that the phenomena is better explained by the conception of free electrons chasing one another from molecule to molecule of nerve tissue,—as physicists may teach;—then, it is pertinent to inquire, is this motion transformed into feeling and thought, or is it transmitted along centrifugal neural transit lines to appear in molar motion, as in the voluntary movement of an arm, or in altered or augmented secretion or other forms of molecular motion? Or may it be that part of the afferent motion is absorbed in feeling and thought and part directly transmitted along efferent neural lines?

We are told that the last hypothesis was endorsed by Herbert Spencer in the first edition of his "First Principles," but later in life his views changed, and attention was called to the change in the preface of the last edition of that work; his later conclusion being that thought and feeling were outside the neural transit circuit and could not be equivalent to, although concomitant with, the molecular motion of matter. He had come to believe with duBois-Raymond[13] that, "if we possessed the same knowledge of atoms that we have of the motions of the heavenly bodies we would understand all the phenomena of the physical world, but we would not understand how consciousness arises, nor how the simplest psychical phenomenon comes to be." "It would be," as duBois-Raymond expresses it, "of unbounded interest, if, with our mental eye turned in-

ward, we could observe the cerebral mechanics of an arithmetical problem; or if we could know what dance of the atoms of carbon, hydrogen, nitrogen, oxygen and phosphorus corresponds to the delight of musical sensation, what whirl of such atoms to the acme of sense enjoyment, and what molecular storm to the frantic pain from maltreatment of the nervus trigeminus." "But however carefully we might follow the motions of individual atoms in the brain, we could see only motion, collisions, and again motion."

And so it is that we are returned by material paths to seemingly soundless depths where lie hidden the mysteries of energy and consciousness.

According to Mr. Percival Lowell,[14] "the neural current of molecular change passing up the nerves and through the ganglia reaches at last the cortical cells, there to find a set of molecules less accustomed to this special change. The current encounters resistance, and in overcoming this resistance it causes the cells to glow. The white heating of the cells we call consciousness. Consciousness, in short, is probably nerve glow." This, of course, is the mechanical point of view delightfully simple and easily grasped. But admitting that the cortical cells do glow, what and where is the percipient? Do the cortical cells cognize their own glowing? The molecules of a wire glow literally if the wire is not sufficiently large for the free passage of an electric current; yet one would hardly suppose the wire to be conscious because the vibrations of its molecules are timed to produce the kind of ether waves we recognize as light.

The terms "cerebricity" and "neuricity," which have crept into our medical literature, descriptive of the energy supposed to be stored in the cortical cells of the cerebrum and minor nervous ganglia, were born out of wedlock most likely, but they stand for a new idea, and the minds of men run after new ideas in science, as in theology, "even as sparks fly upward."

It seems as if the tendency has been towards reducing all the material sciences to problems in molecular physics and of late certain electro-physicists are claiming the earth with all creatures of the land and sea, — and not these alone, — "the golden sun, the planets, all the infinite hosts of heaven," if we may use the poet's phrase, — are counted subjects and creatures of electricity.

In passing to a consideration of the transmissive brain-function theory, it is embarrassing to remember the contempt which the so-called "deterministic" psychologists and some who are wedded to the liver-bile brain-thought idea hold for any hypothesis which admits the possibility of a non-material intelligent reality that may exist apart from a nervous organization. In the face of such problems, however, as the essence of matter and energy and the origin of life, sensation and consciousness, pride of opinion seems hardly worth while.

Some of us have failed to understand how a nerve cell or a nerve fibre can think, hope, fear, love or aspire in spite of all we have been taught of the brain's wonderful mechanism, and it is quite as difficult to understand how many nerve cells and nerve fibres acting in unison and helping one another can produce a sensation, even, unless we assume some sort of a percipient that can interpret the molecular vibrations and atomic motions which seem to constitute the physical part of feeling and thought.

It is somewhat comforting to learn that men of such scientific attainments as Virchow, Helmholtz, duBois-Raymond, William James, and John Fiske in the full maturity of their magnificent intellects, have, likewise, been unable to understand, from such an hypothesis, how feeling and thought come to be. It does seem to be true, as Schiller[15] and James[16] have pointed out, that we have quite as good reason for thinking of the function of the brain as a transmissive function as to think of it as producing thought and

consciousness as the liver produces bile. As the keys of an organ open successively the pipes and let the wind in the air chest escape in various ways; or as the prism transmits, bends, distorts, or separates into the colors of the spectrum the white light of nature, so the brain is conceived to transmit in part, bend, distort and separate, according to its integrity and plane of development, the infinite intelligence of an Eternal Omniscience. As Prof. Schiller puts it, "Matter is an admirably calculated machinery for regulating, limiting and restraining the consciousness which it encases. If the material encasement be heavy and simple, as in the lower organisms, it permits only a little intelligence to permeate through it. . . . If a man loses consciousness as soon as the brain is injured, it is clearly as good an explanation to say the injury to the brain destroyed the mechanism by which the manifestation of consciousness was made possible, as to say it destroyed the seat of consciousness."

The brain-productive-function and the brain-transmissive-function theories both agree that the phenomena of mind are conditioned by the action of some kind of energy upon cells of living nervous matter; but whether this energy is a minute part of that universal energy which keeps the planets and stars true to their courses, or a special kind of "vital force" belonging alone to living organisms, science does not yet permit us to say.

The production theory assumes that the relationship of conscious intelligence to the brain may be likened to music and the harp. When the harp is broken there can be no music for evermore. The transmission theory may also liken the brain to the harp, and those outward phenomena of mind of which voice, gesture and smile are parts, to those vibrations of ether we recognize as pleasing harmony or unpleasant discord. But the transmission theory takes further notice of the harper, without whom there can be no

music, and the harper is likened to the mind or conscious intelligence, whose instrument is the whole human organism, more especially the brain.

We do not suppose such reflections come within the domain of science; certainly not within the scope of any one of the concrete sciences. If every proposition be entirely true, science could not demonstrate the truth, because the conditions are outside the sphere of man's physical experience, and "science is but the codification of experience and is helpless without the data which experience furnishes."[17]

Must we then apologize for asking your attention at this hour to groups of ideas not strictly relevant to the problems of any concrete physical science? Man seems to be the only animal capable of abstract reasoning, and it may be that the "Absolute Mind" intended man should use his peculiar talent for the gratification of an instinctive interest in his own destiny in time, and after time, for him, has ceased to be.

We have purposely avoided introducing such evidence of the existence of a non-material intelligence as the reports of the societies for psychical research offer in abundance, and have refrained from quoting from such writings as Hudson's "Laws of Psychic Phenomena" and "The Scientific Demonstration of a Future Life," — although we have no reason to suppose the investigations of the "psychical researchers" were not carried through in a scientific spirit. We would consent to Hudson's proposition that "a mental state is as much a fact as a mountain," but the field is too broad and the details too lengthy to be of service now. If any fellow would examine this line of evidence, we would refer him to Frederick Myer's "Human Personality," published by Longmans, Green & Co., of New York.

Some members of this Society were studying psychology in a practical way before the reader had learned even the names of the cranial nerves. At the bedside of suffering

mothers you have helped into the world tiny beings with no self-consciousness and for whom world-consciousness was yet but dawning. The manifestations of love and aversion, hope and fear, joy and sorrow, anticipations, aspirations and alternating states of consciousness and unconsciousness are very familiar phenomena to you. If you have not measured, in time, different cerebral reactions nor constructed theorems, or classified, — as have the professional psychologists, — yet, more directly, you may have reached conclusions as substantial as theirs.

Attraction plus repulsion of material molecules may solve the riddle of life to your satisfaction. You may believe that thought and feeling are as much modes of motion of matter as are heat, light or electricity. You may hold that "to know and to feel and to will" are but varying kinds or degrees of excitement of a nervous organism, — nothing more and nothing less. If you are forced from this position by the demonstrations of science that every unit of afferent energy and every bit of centripetal motion can be accounted for by an equivalent quantity of other forms of efferent motion, molecular or sensible, without including conscious intelligence in the circuit at all, — then you may fall back with Ernst Hæckel to the obscure, if less vulnerable position, that the germs of mind are and have been inherent attributes of matter from before the time when this earth, — a gaseous cloud of nebulous matter, — was a new born child of the sun.

On the other hand, giving due weight to the unvarying concomitance, within the limits of our experience, of the phenomena of mind with neural undulations and molecular activity of the body, you may yet hold that the relation, so far as science can reveal, is but a concomitance or induction of material motion by an unknown energy.

If we allow our minds a wide range and notice with F. C. Schiller, William James and John Fiske all the testi-

mony the concrete sciences have to offer, which our intelligence will accept as pertinent; — then I venture the opinion that many of us will be forced to conclude that the transmission theory of the relation of psychic phenomena to the brain is the theory most consistent with all the facts and most worthy the credence of scientific men.

But whatever conclusion we reach the imperfections of science limit us to a conclusion based on moral probability. We assume that members of this Society are not yet ready to accept the credentials of a moral and mental science. "Nevertheless," in the language of Sir Oliver Lodge,[18] "many of us are impressed with the conviction that everything in the universe may become intelligible if we go the right way to work." And so it is that every new discovery or theory in molecular physics, or in any science that may be reduced to electro-physics, may appeal, not alone to our professional interests, — but to the very instincts that are the heritage of our common humanity.

For many years men of science have accepted the hypothesis of an immaterial cosmic ether pervading all interstellar as well as all intermolecular space. Science has insisted on the right to this assumption, in order to frame a partial explanation of such a common phenomenon as light.

For many years the unscientific world has accepted the hypothesis of an immaterial entity called spirit or soul. Ordinary sense has insisted on the right to this assumption in order to explain certain phenomena common to conscious mental life.

Some men have doubted the existence of an imponderable entity like cosmic ether which cannot be seen, heard, felt, tasted or detected by the reagents of the chemist, yet these doubters have offered no explanation of common physical phenomena, independent of this hypothesis, that would be accepted by science or common sense.

Forty years ago it was an easy thing for newly fledged scientists to explain the phenomena of conscious intelligence to their own satisfaction, without the hypothesis of soul or spirit. Today it is safe to say a majority of those well versed in science will agree with Sir Oliver Lodge that "Testimony is borne to inner personal experience on which physical science does well to be silent." But physical science has made wonderful strides in the past decade, and we must admit that molecular and electro-physics have carried off most of the honors. Roentgen's epoch-making discovery in 1895 put the student mind on the *qui vive*, and since the discoveries of Becquerel in 1896 and the Curies in 1898 radio-activity has easily held the centre of the scientific field.

The mental processes and verifying experiments by which the cathode rays and the resulting "X-Ray" phenomena and the emanations of radio-active bodies have been traced back to elementary electric units, of opposite signs, are complex enough and may not be of practical importance to physicians. But to follow these experiments gives us a peep at some of Nature's guarded secrets and may be worth while. Some of us have thought of Nature in a general way as a composite of matter and spirit. We may have loosely thought of electricity as a substitute for the spirit element of the composite. At least we have often heard it claimed that vital processes are electric processes and neural currents are electric currents. If we accept the electronic theory, we must now think of ponderable matter as a form of electricity, while the essential nature of vitality and mind is as obscure as ever. The more we think of it the more this idea seems to harmonize with our observation of electric phenomena.

Whenever man has attempted to apply electricity to the wheels of industry his success has been signal; whenever he has attempted to apply electricity to the destruction of

abnormal and parasitic life, his succcess has, at least, been partial; but whenever he has attempted to reinforce by electric energy, so-called, that energy we think of as vital, —we can almost as truly say that his failure has been signal.

We do not forget that a flagging heart may be stung into temporary activity by a Faradic shock,—a sensitive nerve vibrating pain may be soothed by Galvanism and metabolism, may be influenced indirectly by static and high-frequency currents. But admitting this, other agents may serve as well to whip up a flagging heart, and the forceful impaction of hot and cold water may, indirectly, influence metabolism and modify nerve currents quite as surely. Indeed it seems as if the wonderful fruits of recent scientific investigation had not added greatly to the physician's armamentarium. The more light we get the more it seems to be true that nature was kind enough to furnish man her richest healing agents,—water, fresh air and sunshine,— before he had learned the alphabet of science.

May we requote, "Electricity is not a form of energy any more than water is a form of energy. Water may be a vehicle of energy when at a high level or in motion; so may electricity."

Again it seems as if Nature had hidden from man and denied to his control life destroying agents, among which electricity in motion may be counted, until he has reached, at least, the second grade in the school of science.

The phenomena of the X-ray is now fairly familiar to the most of us. So familiar indeed that hardly a thrill of awe is felt as we look through flesh and blood to confirm or disprove a diagnosis about organs deeply covered and beyond the reach of any hitherto known light.

By the aid of that cleverly designed mechanism, the spinthariscope, we have watched the emanations of a minute particle of a radium compound, less than a hundredth of a

grain, perhaps, and yet its seemingly exhaustless energy, its diamond-like brilliancy and the measure of its rhythmical scintillations made it seem as if it were looking at a microscopic reproduction of the great dog-star Sirius,—most conspicuous among "fixed stars," that glorify a winter's night. Our interest in the phenomena of this microscopic portion of nature is not alone because of its visual similitude to the grandest phenomena of the inorganic universe but more to the significance of the testimony it bears to new truths concerning Nature of which we had not dreamed.

If it be true that radium, uranium and thorium are as surely elements as are bromine, lead and gold, and if it be further true, as seems proven, that these elements are being spontaneously transmuted into helium and other elements not yet identified,[19] then the whole theory of the indivisible atom and the unchangeable element, upon which so many scientific edifices have been builded, must, perforce, be discarded as misleading and outgrown.

If it be proved beyond question that matter may be reduced to electric monads, corpuscles or electrons, each having a "mass" or "weight" one eight-hundredth and a bulk one hundred-thousandth that of an atom of hydrogen, and if we must think of the hydrogen atom as an aggregation of perhaps eight hundred electrons and the nitrogen atom with fourteen times as many, and the iron atom with fifty-six times as many, and each element with such a multiple of eight hundred electrons as corresponds with its atomic weight,[20] then, indeed, does the essential nature of electricity become a question of fascinating interest.

If we accept the theory that radio-activity is due to the explosion of atoms through the fierce energy of electrons within and think of these electric monads as mostly negatively charged, "flying about, each of them repelling every other, but all attracted and kept in their orbits by the mass

of positive electricity in which they are embedded," imprisoned within the limits of a material atom,—then we may better understand how the degree of radio-activity and atomic weight of a substance will be relatively small or large according to the number of electrons of which its individual atoms are composed.

If it prove true that radio-activity of elements and their compounds vary with the atomic weight—and atomic weight is determined by the number of electrons per atom—then we might, *a priori*, except animal organisms to be radio-active in a degree corresponding to those radio-active elements which enter into its composition. Experiments seem to show that certain animals are radio-active; but here the intensity of the radio-activity seems to vary with the vital activity of the animal; for we are informed by Lommasina[21] that the intensity of the radiation, or emission, is greater in grown birds than in young ones and greater in moving individuals than in those at rest.

And so it is that "bio-radio-activity calls for our attention. The call is low and uncertain, as yet, but it is a call to which our professional ears are better attuned. When nerve fibre and brain cells are found to register on calcium phosphide screens or specially prepared photographic plates[22] activities to which the human ear is deaf and to which the eye has hitherto been blind,—then we may feel to say to the enthusiastic physicist, "this throws new light on physiology and is really worth while."

If members of this Society were to select a single name representative of American intellect and scholarship from among the dead who live in our memory, to be written on the roll of fame with the best names of the old world, I fancy Oliver Wendell Holmes and John Fiske would each have some of your votes. Not a few of you knew the bright and genial "Autocrat" personally. Some of us who never looked upon his face loved him hardly less and

almost envy you who were privileged to know him well. While the ripple of his graceful poetry and prose may hide the steady current of his scientific thinking, you know how few there were better conversant than he with the worth and the limitations of science. You know, too, how suggestive are some of his pages of a prescience of realities outside the boundaries of science proper,—as if his soul had caught wireless messages concerning realities beyond the range of our imperfect organs of sense.

If the poetic temperament of Dr. Holmes would rank him among those kindred spirits whom Dr. Osler,[23] with apt metaphor, has called "the Teresians," those who see visions and dream dreams and walk not alone by sight,—John Fiske truly must be classed with a different temperamental groupe. Not to the Laodiceans, surely, for we cannot conceive of the Cambridge philosopher as satisfied to "get and to beget," merely, nor could we think of his being lukewarm about any questions pertinent to "The Destiny of Man." His was pre-eminently the judicial type of mind. For many years in close touch with the best intellects of your own University at Cambridge, and not these alone, a graduate of the department of law; well balanced by nature; thoroughly trained in the laws of evidence, he applied himself diligently to collecting and correlating evidence, not alone of historical and political facts, but of facts from each of the physical sciences which, woven together, vastly enlarge our conception of that Universe of which we are part.

If, then, we may further use the figure of Dr. Osler, John Fiske was a "Gallionion" by temperament and a scientist and historian by training. He accepted without reserve the essentials of the scheme of evolution as outlined by Darwin and elaborated by Spencer and others. His interest was intensely alert to the findings of science in the search for physical realities. He would have agreed with

Lord Balfour[24] that "sense perceptions supply the premises from which we draw all our knowledge of the physical world. What we see depends not merely upon what is to be seen, but on our eyes. What we hear depends not merely on what there is to hear, but on our ears. Now eyes and ears have, as we know, been evolved in us and our brute progenitors by the slow operation of natural selection. And what is true of sense perception is, of course, also true of the intellectual powers which enable us to erect upon the frail and narrow platform which sense-perception provides the proud fabric of the sciences."

We dare to say that John Fiske would have endorsed the teaching of Lord Balfour because many of the truths forced home by the eloquent Chancellor of the University of Edinburgh had in different words and at different times been given to the world by the clear-headed teacher at Cambridge, Massachusetts.

Lord Balfour's magnificent address to the British Association for the advancement of science at once fires our imagination and deepens our reverence. It cannot be otherwise. We catch the ring of scientific truth. We know the Premier's standing in the scientific world. We know by reputation the men whose experimental researches furnish his evidence. In the light of his marshalled facts we see, with the mind's eye, an all pervading space-filling ether which is not atmosphere or a gas, nor does it possess any of the properties of ordinary matter. Seemingly as intangible as the "mind stuff" of the older metaphysicians, we see this hypothetical matrix give birth to and support countless millions of electric monads, which group into atoms of matter. Again we see, mentally, these numberless atoms "concentrated into nebulæ, into suns, and all the hosts of heaven." We see "how at least in one small planet they combined to form organic compounds; how organic compounds became living things; how living things,—

developing along many different lines,—gave birth at last to one superior race; how from this race arose, after many ages, a learned handful, who looked round on the world which thus brought them into being, and judged it and knew it for what it was."

This is a partial epitome of the story of evolution so far as science has cut the leaves. Under the spell of Lord Balfour's eloquence it almost seems as if the whole story was told. Out of something non-material, in the ordinary sense, science now postulates the creation of all material things. Emphasis is deservingly laid on the fact that our organs of sense, so well adapted to the lower planes of evolution, may limit as well as disclose realities which are and may forever be. Arthur Balfour does not claim even to outline the whole story. Indeed, he outlines what science does teach to make clearer, what true science does not claim to explain. "One thing, at least, will remain, of which this long-drawn sequence of cause and effect gives no satisfying explanation," continues Lord Balfour, "and that is knowledge itself." Even as science must postulate an all-pervading entity called cosmic-ether to explain the most familiar phenomena of Nature;—so must philosophy postulate a cognizing percipient called mind, without which all this molecular and inter-molecular motion, which is the physical representation of life, could not be understood nor yet perceived. In the light of all the known facts does it seem incredible to you that Mind should be the objective towards which all the processes of evolution in Nature have been working through past æons? Does it seem a thing incredible that these organs of sense,—even now less acute than those of animals on a lower evolutionary plane,—should, in the further evolution of mentality, become less and less necessary to human personality?

I do not forget that my hearers are busy men chiefly concerned in the science and art of medicine. These facts

and fancies are somewhat foreign to our every day work. And yet we know that underneath the professional current of many a busy physician's life are quiet depths where the testimony of Nature in all her phases are ever welcome. This has been my hope, and, if needful, this must be my apology. It has been my effort,—not to bring you a new or strange message;—rather to gather from scientific fields and speculative by-ways such flowers of fact and fancy as seem most true and beautiful and most suggestive, when woven together, of the sublimity of Nature and the glory of that Eternal Energy, which, in the language of John Fiske, "manifests itself to our consciousness, in harmonious activity throughout the length and breadth and depth of the universe, which guides the stars for countless ages in paths that never err and which animates the molecules of the dew-drop that gleams for a brief hour on the shaven lawn,—whose workings are so resistless that we have naught to do but reverently obey them, yet so infallible that we place our trust in them yesterday, to-day and forever."

REFERENCES.

1. "Life Everlasting," page 65.
2. Wm. James, Psychology, page 102.
3. American Journal of Insanity, April, 1904.
4. "The Riddles of the Sphinx."
5. "Human Immortality."
6. "A Modern Symposium," page 24.
7–8. General Physiology, page 136.
9. Ibid, page 104.
10. Ibid, page 307.
11–12. Ibid, pages 304 and 305.
13. General Physiology, Verworn.
14. "Occult Japan," Boston, 1895.
15. "Riddles of the Sphinx," F. C. S. Schiller.
16. "Human Immortality," Wm. James.
17. "A Crumb for the Modern Symposium." John Fiske.
18. Contemporary Review, Dec., 1904.
19. "Modern Alchemy," Prof. R. K. Duncan, in Outlook, 2–25–'05.
20. "Modern Theory of Physical Phenomena," by Augusto Righi, translated by Trowbridge, and "The Electronic Theory of Matter," Scientific American Supplement, Nos. 1428 to 1434, and "Modern Alchemy," Prof. R. K. Duncan, Outlook, 2–25–'05.
21. Comment on Dr. Th. Lommasina's recent communication to French Academy of Science, Scientific American Supplement, February 4, 1905.
22. Scientific American Supplement, July 30, '04, and June 3, '05.
23. "Science and Immortality," Ingersoll Lecture, Dr. Wm. Osler.
24. Address to the British Association for the Advancement of Science, by the Right Honorable A. J. Balfour, Chancellor of the University of Edinburgh, Scientific American Supplement, No. 1497.

ARTICLE II.

THE SHATTUCK LECTURE.

SOME PROBLEMS OF INTERMEDIARY METABOLISM.

By RUSSELL H. CHITTENDEN, Ph.D.
OF NEW HAVEN, CONN.

DELIVERED JUNE 13, 1905.

SOME PROBLEMS OF INTERMEDIARY METABOLISM.

It requires little imagination and little knowledge of physiology to comprehend that upon a true understanding and rightful interpretation of the many and varied metabolic phenomena of the body depends in large measure the accuracy of our knowledge of the processes of life, both in health and in disease. Yet as Sir Michael Foster has clearly pointed out, it is wholly impossible at present to make any connected or continuous story of the metabolic changes undergone by the individual constituents of the food, the body or the waste products. In the chain of events from the assimilation of the digested food materials by the hungry tissues down to the elimination of the final waste products by the kidneys, lungs and skin, are many broken and missing links by which the continuity of the story is destroyed and we are compelled to fill the gaps by guesses and conjectures, too often widely divergent from the truth.

The very existence of these gaps and breaks in our knowledge, however, serves as an ever-present stimulus inciting renewed activity in the search for truth and enlightenment. As is often the case, this search brings added complexity by opening up vistas that were not supposed to exist, and our thoughts and our experiments take a new turn, from time to time, as additional facts are brought to the surface, with a suggestion of new methods of interpretation. Advances are constantly being made; old views

are undergoing modifications or are entirely supplanted by new theories, the result of steady progress in knowledge. The gaps and breaks in the chain of events that represents the processes of life are being slowly filled in, but new gaps make their appearance, demanding additional knowledge before we can hope for a perfect understanding of the many and varied steps in the metabolic transformations characteristic of living tissues.

As has been so clearly expressed by many writers, we need full knowledge of *all* the steps in the building up and breaking down of the tissue materials, with full consciousness of the fact that a large majority of the diseases of the body are the result of a perversion of metabolism. Further, we realize full well that it is equally important to possess knowledge of *where* these individual steps in metabolism are located, if we are to have full comprehension of the true functional activity of the individual organs and tissues of the body. We need to know the exact nature of each link in the chain of events, and where each link lies with reference to the other links of the chain. The many degenerative changes that check the activities of man at a time when he should be in the full vigor of life, we frequently say are due to the disorders of metabolism or nutrition, recognizing perhaps full well the general character of the perversion, but with no adequate conception of exactly what has occurred or what should be done to remedy or prevent. We lack knowledge of the intermediary steps in metabolism, in health and in disease, and if we are to interpret aright the conditions that bring about disease, to say nothing of prevention and cure, we must acquire full understanding of the processes of intermediary metabolism, their relationship to each other, their general and specific character and the influences that control their harmonious working.

As has just been stated, many of the gaps and breaks in

our knowledge of metabolism have been filled up, new facts have been brought to light, new suggestions have been made, and our knowledge has been correspondingly increased. It has therefore seemed to me that I could not better fulfil the purpose of the Shattuck lecture than to present to you some of these problems of intermediary metabolism which of late have been under consideration and for which we have now a partial or complete solution.

Every active tissue of the body, every glandular organ, every individual living cell, is the seat of incessant chemical activity; construction and destruction are going on side by side, new food materials — proteids, fats and carbohydrates, together with inorganic salts, water and oxygen — are continually being supplied, and passing into the cells of the various tissues or organs are eventually built up into the tissue material, or are made more or less completely adherent. Simultaneously, the cell substance or the adherent materials are undergoing characteristic changes. Secreting gland cells are manufacturing enzyme antecedents and storing them up in the cells for future use, or are perhaps pouring the products forth in slightly altered form to give character to the glandular secretion. Other gland cells are elaborating specific products, such as bile acids, glycogen, amido-acids of various kinds, internal secretions to be re-absorbed by the blood and serve some purpose elsewhere in the body; decompositions of great variety are being carried on, and complex substances are being broken down into simpler with liberation of energy, manifest in the form of heat or mechanical work. And so we have from every group of individualized cells in the body a more or less continuous stream of katabolic products, representatives of the destructive changes taking place in the cells of the individual tissues or organs. Further complexity is introduced by the fact that a given set of cells may show many different lines of activity. Thus, the hepatic cells

not only manufacture bile acids and bile pigments, but they likewise produce glycogen from sugar and transform sugar into glycogen, they form urea from various amido-acids and ammonia, they bring about the construction and destruction of uric acid, they decompose the hæmoglobin of the blood and withdraw the iron from the hæmatin molecule to manufacture bilirubin, they bring about combinations between sulphuric acid and certain aromatic substances such as phenol; in a word, they are active in carrying forward a great variety of divergent processes in which oxidation, hydration and dehydration are equally conspicuous.

These processes of metabolism may be viewed from the standpoint of ultimate changes, without regard to the few or many intermediary steps in the process. Thus, the physiologist may study, for example, the extent to which the formation of urea goes on in the body under different conditions of diet, etc., and he may acquire much useful information regarding the rate of proteid katabolism, without, however, learning much or anything of the various intermediary steps in the process by which the urea is elaborated from the proteid of the food or of the tissues. It is obvious that we can acquire full and exact knowledge of the way in which urea is formed only by searching out the successive changes that take place from the primary breaking down of the proteid to the ultimate formation of urea; *i. e.*, by studying the intermediary processes of proteid katabolism.

In any consideration of intermediary metabolism, where of necessity the action of specialized groups of cells—as in individual tissues and organs—is involved, we are at once confronted with the general question as to how the processes of destructive decomposition are accomplished. In what manner and by what agencies are the processes of katabolism carried out? Until recent times we have had no logical answer to this question. Physiologists and biolo-

gists long held that the processes going on in living tissues were of far too complicated a nature to admit of explanation by chemical or physical means. Schönbein, for example, maintained that the exact sciences were not sufficiently developed to render possible any exact analysis of the changes taking place in living cells. It was the fashion to assume that it was the functional activity of the living cell as a whole that made possible the chemical changes characteristic of the tissue or gland. Cell activity was considered as the result of an inherent quality of the living protoplasm, and no one agent or definite substance could be held responsible for this activity. The theories of vitalism held the physiologist in a strong grasp. Even when ferments or enzymes gained gradual recognition, their action was considered as purely extra-cellular, and they were not looked upon as playing any special part in metabolism. In digestion, of course, their action was manifest, but it was wholly extra-cellular, though their origin was intra-cellular.

Living cells produced enzymes for certain purposes, but there was no evidence that the ordinary processes of katabolism with which the cells might be involved were connected in any way with ferment or enzyme action. Until very recent times, the yeast cell was taken as a typical example of the then point of view. It produced an inverting ferment which could be extracted from the cell and which was very efficient in inverting cane sugar, but the peculiar property of the yeast cell, *i. e.*, the formation of carbon dioxide and alcohol, was the result of the activity of the cell as a whole. It was considered as due to an inherent quality of the living protoplasm. This view prevailed until a few years ago, when Hans Buchner showed that it was quite possible to press out of the yeast cell a clear fluid which, freed from all cellular elements, would still cause alcoholic fermentation. Still later, an enzyme was isolated —zymase—which breaks down sugar into alcohol and

carbon dioxide quite as effectively as the yeast cell itself. In other words, we have learned that this so-called vital property of the yeast cell is due entirely to an intra-cellular ferment or enzyme which the cell manufactures.

In the animal body the processes of metabolism, as judged from the number and variety of products formed, are many and varied. It has been customary to classify these chemical processes under the heads of hydrolytic cleavage, oxidation, reduction, synthesis, etc., and to-day we are in a position to affirm that intra-cellular ferments are the responsible agents in bringing about these varied processes of metabolism in the individual organs and tissues of the body. There is practically no process of metabolism so intricate or obscure that it cannot well be explained by the action and interaction of intra-cellular ferments. New ferments are constantly being discovered, new chemical reactions are being traced to the power of special ferments, and we now speak with great detail of the various *autolytic* changes that different glands and organs undergo, when simply warmed at 40°C. under antiseptic precautions, because of the action of intra-cellular ferments which are practically present in all tissues.

Proteolytic ferments of the trypsin, or of the newly discovered erepsin, type are present in most, if not all, of the tissues of the body. Muscle, liver, kidneys, lymph glands, lungs, spleen, etc., all contain proteid-dissolving ferments, and when the tissues are subjected to auto-digestion or autolysis, such products as the amido-acids, leucin and tyrosin, tryptophan, glycocoll, hexone bases or diamino-acids, and ammonia result from the breaking down of the various proteids of the tissue. In this connection it is to be noted that the respective proteids of the individual tissues are much more quickly affected by these ferments than are foreign proteids, thus suggesting that these intra-cellular enzymes are somewhat different in nature from the ordinary

proteolytic enzymes of the digestive juices. Further, there are peculiar ferments that act specifically upon the intracellular nucleoproteids, splitting off the nucleic acid complex from the proteid part of the molecule, and still other ferments that bring about a cleavage of the nucleic acid with liberation of the contained purin bases. Lastly, it may be said that the general trend of action with these intra-cellular proteolytic ferments is hydrolytic cleavage, much the same as the influence exerted by mineral acids or by ordinary digestive enzymes.

Other types of intra-cellular ferments exercising hydrolytic action accompanied by cleavage are to be found in lipases, true adipolytic ferments which split neutral fats into glycerin and fatty acids in a fashion quite analogous to the action of ordinary steapsin. These lipases are found in the liver and other organs. Further, the kidneys and liver contain a ferment capable of splitting hippuric acid into benzoic acid and glycocoll (Schmiedeberg). Again, Jacoby has shown that the liver will yield an extract capable of splitting off amonia from urea, and that the same gland contains a ferment capable of transforming amido-acids into amides. Moreover, the liver and indeed the kidneys as well have the power of splitting up amides; a fact of the utmost importance in intermediary metabolism. Lastly, glycolytic action, in which both cleavage and oxidation of sugars may be involved, is likewise a function of many cells, and adds another striking illustration of intracellular ferment action, by which carbohydrate metabolism may be carried forward in the different tissues and organs of the body.

Oxidation is preëminently one of Nature's ways of bringing about alteration and decomposition, and in intermediary metabolism especially, oxidative processes must be quite conspicuous. Yet to-day we have accumulated a mass of evidence tending to show that oxidation in the tissues is

due primarily to the presence and action of a row of more or less closely related though chemically distinct ferments, known collectively as oxidases. Physiological oxidation therefore, as it occurs in metabolism, is likewise a result of intra-cellular ferment action. To be sure, the blood is the carrier to the tissue cells of the necessary oxygen, but the process of oxidation, with such chemical reactions as is involved, is due to the action of the intra-cellular oxidases. It is well to recall that Schönbein originally drew attention to the fact that the blood, and tissues as well, had the power when fresh of splitting off oxygen from hydrogen peroxide, but lost the power when heated to 90–100°. This phenomenon, however, is not, as was at one time thought, simply a general ferment indicator, but is due to the action of specific oxidases, some of which can actually be precipitated from the fluids containing them, and moreover differ among themselves as to the degree in which they are distributed among the different tissues and organs of the body. The spleen, liver, pancreas, thymus, muscle, brain and ovaries are noticeably rich in oxidizing ferment. In fact, the physiologist to-day has knowledge of a large number of intra-cellular oxidases, more or less distinct from each other, to some of which distinct names have been given indicative of their special lines of activity, viz., *aldehydase*, a ferment which oxidizes aldehydes to their corresponding acids, and present in the liver, kidneys, testicles, suprarenals, brain, lungs, thymus and salivary glands; *tyrosinase*, a ferment which oxidizes benzol derivatives, especially tyrosin; and *indolphenol-oxidase*, a corresponding ferment present in the pancreas, thymus, spleen, and salivary glands, but absent from the muscle and kidneys.

As can easily be imagined, these facts throw new light upon the methods of intermediary metabolism. They show us that the individual gland and tissue cells are provided

with efficient agents for accomplishing chemical changes of great variety. We are no longer forced to think or speak in vague terms of the somewhat mystical changes that go on in living tissues, but we have acquired sufficient knowledge to understand that the individual cells of the body are provided with intra-cellular ferments of definite character by which the metabolic changes are produced. These newly discovered enzymes are of such widely divergent nature and so broadly distributed among the tissues of the body that they offer an adequate explanation of the many chemical changes that occur in ordinary metabolic processes. Further, when it is remembered that practically all tissue cells contain proteolytic enzymes capable of transforming their own proteids into simpler products, and that in addition they have special enzymes endowed with a variety of powers, we see opening before us simple chemical methods of tracing out the individual steps in the gradual breaking down of the complex molecules of tissue material into the relatively simple bodies which we classify as intermediate and end products of tissue metabolism.

Let us now consider more in detail, and from the standpoint of what has already been said, some specific examples of intermediary metabolism. By the older methods of investigation, the physiologist was compelled to rely mainly for information upon such data as could be obtained by careful study of the urine, reinforced by examination of the individual glands and tissues. The detection in the latter of specific products led to the inference that these substances were formed in the glands under examination, but it was purely a question of inference as to how and from what the bodies originated. Further, if the urine showed the absence of these same bodies, then we naturally inferred that they were either completely destroyed or transformed into some other body, the methods of which were likewise enigmatical. The gaps and guesses of Foster were met with

at every turn in any attempt to trace out systematically the changes of intermediary metabolism.

Modern methods and recent acquirements in knowledge are both well illustrated in what has been accomplished in the study of purin metabolism. Nucleoproteids of various kinds are conspicuous constituents of all cells; they are found in all tissues, in all glandular organs, and their widespread distribution may be taken as evidence of their great physiological importance. Their metabolism must of necessity be a conspicuous feature in the changes taking place in all glandular organs. Chemical study has shown that nucleoproteids by simple hydrolysis with mineral acids in a flask can be broken down into some form of proteid, phosphoric acid and one or more purin bases, such as adenin, guanin, xanthin and hypoxanthin. Further, chemical constitution would lead to the inference, verified by repeated experiment, that these purin bodies can be readily converted into uric acid. Do such reactions take place in the body; and, if so, by what agencies are they brought about?

If such glands as the thymus, suprarenal, spleen, etc., are subjected for some time to self-digestion at body-temperature in the presence of an antiseptic, it can readily be demonstrated that certain chemical changes occur. Thus, in the case of the thymus, large amounts of xanthin and a small amount of hypoxanthin, together with uracil, are found in the fluid. With the suprarenal, large quantities of xanthin and a small quantity of hypoxanthin are found.* With both of these glands, however, guanin and adenin are entirely lacking. In the self-digestion of the spleen, on the other hand, guanin is formed abundantly, also hypoxanthin, while adenin and xanthin are wanting. These results are quite different from what is found when the glands or their respective nucleoproteids are boiled with acid. In other words, when mineral acids are used as the hydro-

* Walter Jones: Ueber die Selbstverdauung von Nucleoproteiden. Zeitschrift für physiologische Chemie, Band 42, p. 34, 1904.

lyzing agents, then the tissue nucleoproteids yield, as a rule, guanin and adenin, but by autolysis xanthin and hypoxanthin are the conspicuous bases.* This difference in the result by hydrolysis with acid and by autolysis is due to the fact that in autolysis the changes induced are owing to the presence of specific intra-cellular ferments,† which have the power of acting upon certain of the purin bodies transforming them into other related substances.

Thus, in the self-digestion of the pancreas in an alkaline medium large amounts of xanthin and hypoxanthin are found as the end products of the autolysis. Guanin and adenin are no doubt also formed, but they are gradually converted into xanthin and hypoxanthin by intra-cellular ferments. If pure guanin itself is placed in a mixture containing finely divided pancreas, with chloroform to prevent putrefaction, and the mixture kept at 40°C. for some time, the guanin is slowly but surely converted into xanthin, until in time it is wholly replaced by the latter base. The ferment or enzyme that brings about this transformation is called *guanase*,‡ and is likewise present in the thymus and adrenals, but is absent from the spleen. A corresponding intra-cellular enzyme, known as *adenase*, and which transforms adenin into hypoxanthin, is likewise present in the thymus, adrenals, pancreas and liver.§ These two ferments are true hydrolyzing agents, the chemical reactions involved being quite simple:

$$\underset{\text{guanin}}{C_5H_5N_5O} + H_2O = \underset{\text{xanthin}}{C_5H_4N_4O_2} + NH_3$$

$$\underset{\text{adenin}}{C_5H_5N_5} + H_2O = \underset{\text{hypoxanthin}}{C_5H_4N_4O} + NH_3$$

In both of these cases it is to be noted that there is not only a taking on of water, under the influence of the enzyme,

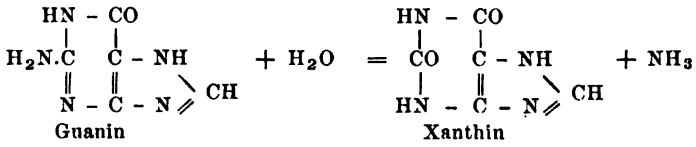
* Compare Levene: The Autolysis of Animal Organs. American Journal of Physiology, Vol. 12, p. 276, 1904.

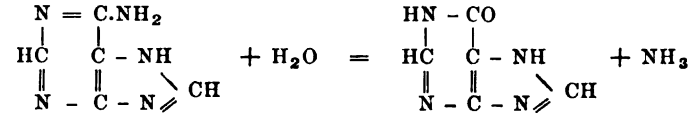
† Jones and Partridge: Ueber die Guanase. Zeitschrift für physiologische Chemie, Band 42, p. 343, 1904.

‡ Jones and Partridge: Loc. cit.

§ See Jones and Winternitz: Ibid., Band 44, p. 1.

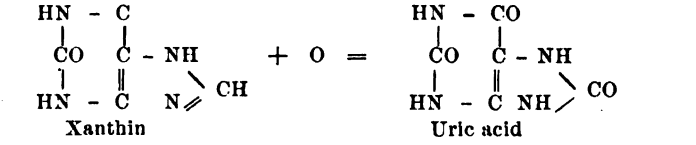
with a retention of the oxygen, but there is also a giving off of ammonia, by which the transformation is made possible. The real essence of the reaction is best shown by use of the constitutional formulæ, whereby can be seen the full extent of the change which is effected:





Expressed in different language, these enzymes are able to transform the two amino-purins into the corresponding oxypurins xanthin and hypoxanthin; *i. e.*, they are typical deamidizing ferments, destroying the NH_2 group of the adenin and guanin.

Again, it is possible, as Schittenhelm* has shown, to prepare from the spleen simple extracts, free from all tissue elements, that are capable of transforming these purin bodies into uric acid. From such extracts, the enzyme which is the active agent in the oxidation can be separated by simple chemical means, and the purin bases can be transformed quantitatively into uric acid by its action. The conversion of xanthin, for example, into uric acid is purely a process of oxidation brought about by a typical intra-cellular oxidase, the reaction being as follows:



* Ueber die Ferments des Nucleinstoffwechsels. Zeitschrift f. physiologische Chemie, Band 43, p. 228. See also Schittenhelm: Ueber die Harnsäurebildung in Gewebsauszügen. Ibid., Band 42, p. 260.

Schittenhelm* has already demonstrated that this oxidizing enzyme is present in the liver, spleen, lungs and muscle, but is absent from the thymus, intestine, blood and kidneys, and Burian has given to the ferment the name of "xanthinoxidase."

Another interesting fact to be brought forward in this connection is the existence of a specific intra-cellular enzyme appropriately termed *nuclease*, which has the power of liberating the purin bases from their combination as a component part of tissue nucleoproteids, or more specifically of the contained nucleic acids. It has long been known that nucleoproteids, nucleins and nucleic acid when fed cause at once an increased output of uric acid, and it has been clearly recognized that uric acid as a product of metabolism must result from the transformation of the nuclein bases which are so conspicuous in the nucleins and nucleic acid; but as to how the uric acid was formed in the body could only be conjectured. Now, however, the matter is made quite clear, and we see how various intra-cellular enzymes working one after the other are able gradually to evolve uric acid from tissue nucleoproteids. Under the influence of a *nuclease*, nucleic acid is split up with liberation of the free nuclein bases, then by the action of a deamidizing enzyme guanin and adenin are transformed into xanthin and hypoxanthin respectively. Further, by the action of the oxidase just referred to, hypoxanthin is oxidized to xanthin, and the latter is converted into uric acid. Moreover, it is worthy of emphasis that these enzymes are not distributed indiscriminately, but are confined to specific organs or tissues, as has already been indicated. Finally, it is to be noted that there is another tissue oxidase — contained so far as is known at present in the kidneys, liver, muscle, and perhaps the marrow of bones (Schittenhelm) — which has the power of oxidizing and

* Loc. cit. Band 43, p. 236.

thus destroying uric acid. Here, then, we have four distinct enzymes or intra-cellular ferments, more or less responsible agents for the production and presence of uric acid in the body. The various steps in the intermediary metabolism, by which this substance results are made quite clear and we see how specific intra-cellular enzymes, formed by the tissue cells, are responsible for the chemical changes induced.

By all students of metabolism, the two-fold origin of uric acid — endogenous and exogenous — is well understood. It is equally well known that in the origin of uric acid from outside materials, free and combined purin compounds are equally effective; *i. e.*, nuclein-containing foods and nitrogenous extractives of the xanthin type. By the processes of metabolism, in which the intra-cellular enzymes referred to are the active agents, uric acid is formed, and eventually appears in the urine. It is evident, however, that exogenous uric acid cannot be controlled wholly by the character and quantity of the food consumed. To be sure, avoidance of purin-containing foods will necessarily result in a greatly diminished production of uric acid, but we must not overlook the part which the uric acid-oxidizing ferment plays in the destruction of this substance in the tissues of the body. As has already been pointed out, this peculiar oxidase has been found by Schittenhelm in the liver, muscle, kidneys, and perhaps in the bone marrow, and it is easy to see how the content of uric acid in the blood, lymph and urine may be determined as much by the relative activity of this oxidase, as by the activity of the nuclease, amidase and oxidase which produce uric acid from the purin-containing foods. It is perfectly clear that inhibition of the oxidizing action of the uric acid-destroying enzyme may be as effective in bringing about a high content of uric acid as increased production of uric acid. We have no means at present of judging the relative activity, under normal meta-

bolic conditions, of these two somewhat divergent factors, but I believe that the conspicuous increase of uric acid noticed under many conditions of life is due as much or more to increased inhibition of the oxidation of uric acid as to augmented production of uric acid. This is conspicuously true in the influence exerted by alcoholic drinks on the uric acid-content of the blood and urine.

Recent work carried on in the writer's laboratory* has shown quite conclusively that in man the ingestion of alcoholic fluids with purin-containing foods increases at once the output of uric acid in the urine. Alcohol, however, does not produce this result when taken with a light diet or one free from purin compounds. In other words, alcohol influences only the uric acid of exogenous origin, in conformity with the well-known gouty tendencies of high proteid feeding combined with consumption of alcoholic fluids. Alcohol is well understood to interfere with the oxidative processes of the liver, and it is more than probable that its influence on the uric acid content of the blood lies in the direction of inhibiting the action of the oxidase which, present in the liver and other tissues, normally destroys or oxidizes a certain proportion of the uric acid formed. This being true, we see another illustration of so-called perverted metabolism due entirely to a change in the rate of action of an intra-cellular oxidase.

In any reference to the origin of uric acid in the body, too much stress cannot be laid upon the easy convertibility of the *free* purin bases adenin, guanin, hypoxanthin and xanthin into uric acid,† by virtue of the action of the intracellular enzymes present in so many of the organs and tissues. In the ordinary foodstuffs, however, as in meat

*S. P. Beebe: The Effect of Alcohol and Alcoholic Fluids upon the Excretion of Uric Acid in Man. Americal Journal of Physiology, vol. 12, p. 13.

†See Krüger and Schmid: Die Entstehung der Harnsäure aus freien Purinbasen. Zeitschrift f. physiologische Chemie, Band 34, p. 549.

broth, it is the oxypurins hypoxanthin and xanthin that the body has to deal with mainly. These are quickly oxidized to uric acid which may then be excreted, or a portion may undergo oxidation to urea or other products. When, on the other hand, combined purins are introduced, as in the nucleins and nucleoproteids, adenin and guanin are liberated by the action of nuclease. These bodies are amino-purins, and their continuance unchanged depends upon the presence and action of the two enzymes adenase and guanase. If the latter are present and active in normal degree, we can conceive of a ready conversion into hypoxanthin and xanthin and then into uric acid. But if these enzymes are lacking or are inhibited in their action, then the two amino-purins will float about unaltered for a time at least. May we not find in this possibility an explanation for certain disturbed conditions of the body, especially of the kidneys, since experiments have shown that adenin in the case of dogs,* and in rabbits† when larger doses are given, is liable to produce anatomical alterations in the kidneys, particularly of the tubules, accompanied by peculiar deposits in the kidney tissue of spheroliths of uric acid and ammonium urate, which evidently cause irritation and marked alterations of structure. Here, again, we see suggested a threatened perversion of metabolism through possible absence or inhibition of an intra-cellular enzyme.

Uric acid of endogenous origin is as much a product of enzyme action as that which is derived from the purin compounds of the ingested food. It is well known that the elimination of uric acid does not cease during fasting, or with a diet free from purin compounds. The amount, however, is greatly reduced when purin-containing foods

* Minkowski: Untersuchungen zur Physiologie und Pathologie der Harnsäure bei Saugethieren. Archiv f. exper. Pathol. und Pharm., Band 41, p. 375.

† Schittenhelm: Das Verhalten von Adenin und Guanin im thierischen Organismus. Ibid., Band 47, p. 432.

are excluded from the diet. Many investigators* have shown experimentally that there is a certain degree of constancy in the output of uric acid on a non-purin diet, even when there are wide variations in the quality and quantity of the purin-free food. Various careful experiments, covering fairly long periods of time, carried on in our laboratory by Dr. E. W. Rockwood† on men taking only purin-free food, such as milk, wheat bread, butter, cheese, cereal, fruit, etc., showed a daily output of uric acid averaging 0.3—0.4 gram. Further, it was observed, in conformity with the thesis of Burian and Schur, that a given individual shows a certain degree of constancy in the daily excretion of uric acid. In other words, the elimination of endogenous uric acid is constant for each individual; *i. e.*, it is an individual factor dependent perhaps in part upon the weight of the body or the contained tissues and organs. It was a noticeable fact that the results obtained were not influenced in any measurable degree by the extent of muscular activity.

As to the origin of this endogenous uric acid, it has long been taken for granted that it must come in part from the breaking down or metabolism of the nucleoproteids of the tissue cells, by the action of the same agencies that are effective in the formation of exogenous uric acid. There are, however, other possible methods not to be entirely overlooked; viz., the possibility of a synthetical production of the acid in the liver or elsewhere, and also the possibility of some other antecedent than the nucleoproteids of the glandular organs, leucocytes, etc., serving as the direct mother substance in its formation. Knowing the fate of the nucleoproteids of the food and the marked influence

* Burian and Schur: Archiv. f. die gesammte Physiologie, Band 80, p. 241; also, Band 87, p. 239.
Sivén: Skandinavisches Archiv für Physiologie, Band 11, p. 132.
Wiener: Ergebnisse der Physiologie, 1902, i, Part I., p. 355; Ibid., 1903, ii, Part I., p. 377.

† The Elimination of Endogenous Uric Acid. American Journal of Physiology, vol. 12, p. 38.

these exert upon the elimination of uric acid, reinforced by the abundant experimental evidence that both free and combined purin bases when ingested are transformed into uric acid, there has been little question that the endogenous uric acid must be derived from the breaking down of the nucleoproteids of the tissue cells. Indeed, there has been some disposition to take the amount of endogenous uric acid eliminated as a measure of the chemical activity or disintegration of tissue elements. This view, however, has quite recently received a severe shock in the results of some experiments recorded by Burian,* in which he shows quite conclusively that only a very small amount of the endogenous uric acid has its origin in the nucleoproteids of disintegrating tissue cells or leucocytes, the larger part coming from quite a different source, viz., from the purin base hypoxanthin, which is continually being formed as a metabolic product of living muscle tissues. Still, as already stated, muscular work, which naturally means increased muscular metabolism, has not appeared to cause any measurable increase in the output of uric acid. This may be true, according to Burian, with the 24 hours' urine, but it is not the case when the urine is examined in hourly periods, for he finds that after vigorous muscular exercise in the case of fasting individuals, where the hourly excretion of uric acid is fairly constant, there is a marked increase in the output of uric acid through the urine, the maximum excretion showing itself in the second hour after cessation of the work, enduring for an hour or longer, then slowly sinking below the normal. This fall in the rate of excretion thus balances in a measure the temporary increase, and explains why examination of the 24 hours' urine has failed to afford evidence of this peculiar influence.

This observation of Burian renders probable an intimate

* Die Herkunft der endogenen Harnpurine bei Mensch und Saügetier. Zeitschrift f. physiologische Chemie, Band 43, p. 532.

relationship between muscle metabolism, the formation or liberation of a purin base in the muscle, and its consequent conversion into uric acid by the intra-cellular xanthinoxidase. Further, the same investigator has shown that by perfusing a mixture of Ringer's solution and fresh dog's blood through the hind leg of a recently killed dog, the muscle fibres being maintained in a state of rest, the blood will gradually show the presence of uric acid; while if the muscles are stimulated by an induction current for an hour or so, then the circulating blood shows an increase of purin material, and the living muscle tissue itself shows an increased content of hypoxanthin during the period of "work." It is obvious from these statements that the muscle or the blood must contain xanthinoxidase to convert the liberated oxypurin into uric acid. This is indeed true of the muscle, for an extract of dog's muscle, like an extract of the liver or spleen, will transform to uric acid any oxypurin added, thus affording satisfactory proof that the muscle contains a specific intra-cellular oxidase. Further, there is evidence that the muscle also possesses the power of decomposing uric acid.

There is thus opened up a new chapter in purin metabolism, bearing upon the endogenous production of uric acid. In the resting state, muscle is continually giving up to the blood a certain amount of uric acid formed at the expense of the hypoxanthin which originates within its own tissue. The oxidation of the purin base to uric acid is accomplished by the specific oxidase which the muscle itself contains, but, as Burian points out, this enzyme must be so located that the hypoxanthin is converted into uric acid just as it is passing from the muscle fibre into the blood or lymph, since muscle itself never contains any uric acid, only purin base. Further, it is to be understood that a certain amount of the uric acid is at once decomposed by the action of the other oxidizing ferment which destroys this acid. Finally, Burian calls attention to the fact that

since the hypoxanthin-content of the muscles, in spite of the continuous formation and withdrawal of uric acid, remains in general constant, it follows necessarily that muscles during rest are continually forming new hypoxanthin as a normal product of their own metabolism. During activity, the formation of hypoxanthin in muscle is accelerated, while at the same time there is a corresponding increase in the output of the purin body from the muscle, the action of the xanthinoxidase being doubtless stimulated by the local deficiency of oxygen concomitant with the increased muscular activity, and as a final result there is an increased production of uric acid. What proportion of the thus formed uric acid appears in the urine we have at present no definite information, for we do not know in what degree the formed acid is further decomposed.

Assuming the correctness of the views just expressed, it is evident that muscle tissue takes a new position in the body as a uric acid-producing center. The tissue, it is true, does not differ from other tissues or organs as a depot for the specific oxidase, but unlike the other tissues it is constantly producing, both in rest and in activity, an oxypurin ready for direct transformation. In the other uric acid-producing centers, activity is more or less intermittent, being dependent primarily upon the advent in the food of free or combined purin bodies upon which the intra-cellular amidases and oxidases can act. Burian considers, on the strength of his experimental results, that endogenous uric acid owes its origin mainly to these metabolic changes in muscle, and that only a very small proportion of the endogenous acid is derived from the nucleoproteids of the disintegrating tissue cells. Again, experimental evidence* is strongly opposed to the view that there is any synthetical production of uric acid in the animal body; the only way

* Burian: Ueber die oxydative und die vermeintliche synthetische Bildung von Harnsäure in Rinderleberauszug. Zeitschrift f. physiologische Chemie, Band 43, p. 497.

at present known in which the exogenous uric acid can be increased in amount, being feeding with free purin bases, or with nucleoproteid-containing foods in which combined purin bodies are present. In other words, uric acid owes its origin entirely to processes of enzymatic oxidation.

We may advantageously pause here for a moment to consider the possible bearing of these facts upon so-called oxidation within the body. We have seen how in the processes discussed, oxidation is dependent upon specific ferments or enzymes, and we further understand that the intermediary products formed are definite bodies because of the specific nature of the active enzymes, and secondly, because of the chemical nature of the substances acted upon. In other words, oxidation in intermediary metabolism begins to take on the shape of a series of well-defined chemical processes in which chemical constitution and specific enzyme action are the predetermining causes. In the absence of the particular chemical groups, the oxidase is ineffective to bring about oxidation, or given the proper compound or mother substance in the absence of the specific oxidase there is no oxidation. Oxidation in the animal body, therefore, certainly as applied to intermediary metabolism, is no longer to be classed with ordinary combustion processes. We find, on the contrary, a series of orderly chemical processes, each one of which is presided over by an intra-cellular enzyme, specific in its nature in that it is capable of acting only upon bodies having a certain definite composition, and leading invariably to a certain definite result. Further, it is easy to see how perversion or inhibition of intermediary metabolism may be engendered by causes which act primarily upon the cells where the enzymes in question have their origin, or by influences which may bring about changes in the environment surrounding the intra-cellular ferment; for just as the ordinary extra-cellular digestive enzymes are influenced in their ac-

tivity by surrounding conditions, so may the intra-cellular oxidative enzymes feel the effect of changes in their environment.

In enzyme action in general, maximum results are obtained only when the enzyme and the substance to be acted upon bear a certain definite relationship to each other. A proportionate excess of one over the other may modify the rate of action, and where the substance undergoing change — in intermediary oxidation — is in relative excess over the enzyme there would seem suggested the possibility of a perversion of the normal oxidative change. In such a case as this, some intermediary body — perhaps abnormal in nature — might make its appearance. This principle in metabolism is well illustrated by some recent experiments carried on in our laboratory * bearing on the formation and excretion of allantoin in the animal body. Allantoin is an oxidation product of uric acid, having the formula:

$$OC\begin{matrix} NH. & CH. & HN \\ & | & \\ NH. & CO & H_2N \end{matrix}CO$$

It is found in the urine of pregnant women and in the urine of newly born infants. It is likewise excreted in large quantities by dogs after feeding pancreas, thymus, lymphatic glands, or any other material rich in nucleic acid. It also appears in the urine of the dog and cat after feeding relatively large amounts of uric acid. In other words, it is quite evident from the experimental records that in the case of these animals at least the purin bases (and nucleins) may give rise, under some conditions, to allantoin as an intermediate product of their oxidation. Ordinarily, these compounds of the purin type are more completely oxidized in the system, with formation of urea, although, as we have seen, a certain proportion of the nitrogen is excreted

* Mendel, Underhill and White, American Journal of Physiology, Vol. 8, p. 380. In this article are contained references to a large number of papers bearing on this subject.

as uric acid. But when larger quantities per kilo of body-weight of uric acid, or of the antecedent purin compounds, are introduced or formed in the system, the oxidative enzymes are unable to bring about as complete a decomposition of the purin radicles, and under such conditions allantoin escapes as an end-product of the incomplete oxidation of the acid.

Drugs that are known to influence certain lines of metabolic activity in which the purin bodies are concerned, may well have their action explained by assuming an influence upon enzymatic oxidation, as well as by attributing the effect to some general disturbance of functional activity of the gland or tissues. Further, with this mode of explanation at hand, it is easier to understand how drugs that bring about marked pathological conditions may still be free from influence upon some of the lines of functional activity with which the gland or tissue is associated. Thus, as some experiments recently carried on in our laboratory show,* phosphorus which produces such marked hepatic degeneration does not interfere in the case of the dog with the power to elaborate uric acid during a diet rich in purin compounds. In other words, the specific nuclease, amidase and xanthinoxidase are not inhibited in their action by the presence or influence of phosphorus, but uric acid is formed as under normal conditions when purin-containing foods are exhibited. Further, other experiments† made in our laboratory with sulphonal, a typical hepatic drug, tend to show that in fasting dogs there is no undue formation of allantoin when soluble urates are injected into the circulation, *i. e.*, the normal uric acid oxidation is not interfered with, even though the animal be strongly under the in-

* Mendel and Jackson: American Journal of Physiology, Vol. 4, p. 168.

† Mendel and White: On the Intermediary Metabolism of the Purin Bodies: The Production of Allantoin in the Animal Body. American Journal of Physiology, Vol. 12, p. 93.

fluence of the drug. When, however, animals fed on pancreas, *i. e.*, with an abundance of nucleoproteid, are treated with sulphonal, then it is found that allantoin, which on the above diet would normally appear in large amounts, is never present in the urine. Presumably, the sulphonal in some way influences favorably the oxidation of the purin compounds to uric acid and urea, and it is reasonable to assume that this action is due to some stimulating influence upon the oxidases of the liver or other glands, as to ascribe it to any other more general cause.

We must not, however, confine our attention too long to one particular line of intermediary metabolism, remembering that there are many other chapters in nutrition to which attention may profitably be directed, in illustration of our main theme. Let us therefore consider next some phases of the intermediary metabolism of carbohydrates and its perversions, which will afford us opportunity also to discuss the formation of oxalic acid, glycuronic acid, etc. It is well understood that under ordinary conditions of life and health, the carbohydrates of our food and body tissues are continually undergoing oxidation with ultimate formation of carbon dioxide and water, but it is not to be supposed that this oxidation necessarily takes place wholly at one step. In the blood, as Lépine* originally pointed out, there is present a glycolytic ferment or enzyme which has the power of destroying sugar, but as Krauss† and Arthus‡ have shown, this action of the blood is not sufficiently strong to bring about the oxidation of the several hundred grams of sugar that are being broken down in the body each day. The discovery by v. Mering§ and Minkowski that extirpation of the pancreas produces a severe form of diabetes led

* Comptes rendu, Tome 110, p. 742 and p. 1314.
† Zeitschr. f. klin. Med., Band 21, p. 315.
‡ Arch. de Physiol., Tome 24, p. 337.
§ Diabetes mellitus nach Pankreasextirpation. Archiv f. exper. Pathol. u. Pharm., Band 26, p. 371.

naturally to the view that this organ normally produces something, possibly an internal secretion, failure of which renders the body unable to burn sugar. Search for the presence of a glycolytic ferment in the pancreas, however, gave negative results.* It remained for Cohnheim† to show that when the pancreas is combined with muscle there is developed a strong sugar-decomposing action. The pancreas alone has no such power, and muscle likewise when taken by itself shows no appreciable glycolytic action, but the two together have such power that 5.6 grams of sugar can be destroyed or burned by a kilogram of muscle. If, says Cohnheim, we estimate that an adult man possesses 40 kilograms of muscle, this tissue influenced by the action of the pancreas is quite capable of oxidizing over 200 grams of dextrose.

This oxidizing power is not dependent upon the presence of the tissue cells, but the active agent can be extracted and the resultant cell-free fluid shows the same power of oxidizing dextrose, thus signifying quite clearly that the agent is a soluble enzyme or ferment, which presumably resides in the muscle tissue in an antecedent form and is activated through some influence exerted by the pancreas. The action is thus analogous to the influence exerted by the *enterokinase* of the intestinal glands, which has the power of transforming inactive trypsinogen into the active enzyme trypsin, the proteolytic enzyme of the pancreatic juice. It is true that these results of Cohnheim have been adversely criticised by some investigators, notably by Stoklasa and by Claus and Embden, but nevertheless the general trend of the results seems pretty clearly established, and it appears evident that glycolytic action, which undoubt-

* Herzog: Liefert das Pankreas ein Dextrose spaltendes, alkohol und kohlensäure bildendes Enzym? Hofmeister's Beiträge zur chemischen Physiologie und Pathologie, Band 2, p. 102.

† Die Kohlehydrateverbrennung in den Muskeln und ihre Beeinflussung durch das Pankreas. Zeitschrift für physiologische Chemie, Band 39, p. 336. Also, Band 42, p. 401, and Band 43, p. 547.

edly occurs in most, if not all, of the active tissues of the body, is due to soluble enzymes, the activity of which is heightened through the influence of some agent or agents furnished by the pancreas.

In this oxidation of sugar by the tissues of the body, a large proportion of the sugar burned is unquestionably entirely destroyed, but not always is the oxidation complete, and then intermediate products make their appearance as a sign and a warning of physiological processes gone astray. Incomplete or improper burning of sugar may — in the absence of any better explanation — be ascribed to lack of the specific enzyme or enzymes, or to lack of suitable conditions for their proper activation.

The sugar of the blood, it will be remembered, is dextrose, an aldehyde, having the formula:

CH_2 (OH). CH (OH). CH (OH). CH (OH). CH (OH). CHO

By mild oxidation, the aldehyde group (CHO) is changed into an acid group (carboxyl group, COOH) and monobasic gluconic acid results, viz.:

CH_2 (OH). CH (OH). CH (OH). CH (OH). CH (OH). COOH

By more energetic oxidation, the dibasic saccharic acid is formed, viz.:

COOH. CH (OH). CH (OH). CH (OH). CH (OH). COOH.

These two illustrations suffice to show the readiness with which aldehyde groups (CHO) and primary alcohol groups (CH_2OH) are oxidized to acid groups (COOH). It might be assumed therefore that all substances containing the above groups would undergo oxidation in the body along the lines indicated, just as ordinary alcohol when introduced into the system may yield aldehyde and acetic acid when the oxidation is somewhat restricted. As we have tried to point out, however, oxidation in the animal body is something more than a mere exposure to oxygen, and it does not necessarily follow that all substances con-

taining these groups will be oxidized in rhythmic fashion in the body, or through a regular series of successive stages. The very fact that such results do not always occur affords the best of evidence that animal oxidation involves other factors than simple oxygen; *i. e.*, is in harmony with the view that oxidation of a given substance depends in large measure upon the presence of specific intra-cellular oxidases which can attack that particular substance. This view is forcibly illustrated by the work of Paul Meyer* with ethylene alcohol, or glycol, and its derivatives.

The oxidation products of glycol that would naturally come under physiological consideration are the following, the relationship of which is indicated by the formulæ:

CH_2OH	COH	COOH	COH	COH	COOH
CH_2OH	CH_2OH	CH_2OH	COH	COOH	COOH
Ethylene glycol.	Glycol aldehyde.	Glycollic acid.	Glyoxyl.	Glyoxylic acid.	Oxalic acid.

It has been found by Pohl† that glycol or ethylene alcohol in the case of the dog can be only partially burned in the body, and that its introduction always gives rise to a large production of oxalic acid. Mayer has found the same to be true in the case of rabbits. It is a noticeable fact, however, as pointed out by Mayer, that when glycol or ethylene alcohol is given to rabbits in moderately large doses, there is a large output of glycollic acid. In fact, it may be stated that under the above conditions about one-fourth of the glycol is oxidized simply to glycollic acid, while the presence of considerable oxalate in the urine demonstrates further oxidation of a portion of the glycol to oxalic acid. With sufficient dosage of glycol, rabbits succumb with severe hæmorrhagic nephritis, the glomeruli being practically occluded with crystals of calcium oxalate

* Experimentelle Beiträge zur Frage des intermediären Stoffwechsels der Kohlehydrate. Zeitschrift f. physiologische Chemie, Band 38, p. 135.

† Ueber den oxidativen Abbau der Fettkörper im thierischen Organismus. Archiv f. exper. Pathol u. Pharm., Band 37.

and the whole section of the kidney tissue showing large masses of this crystalline salt.

The significance of these facts in the present connection lies in the probability, as suggested by Mayer, that this peculiar oxidation by which glycollic and oxalic acids result without any appearance of bodies intermediate between these two, is due first to simple oxidation of the primary alcohol group of glycol to the carboxyl group, thus giving rise to glycollic acid, as follows:

$$\begin{array}{l} CH_2OH \\ | \\ CH_2OH \end{array} \longrightarrow \begin{array}{l} COOH \\ | \\ CH_2OH \end{array}$$

By further oxidation of the so-formed glycollic acid, the remaining primary alcohol group of the acid is similarly attacked with formation of oxalic acid as follows:

$$\begin{array}{l} COOH \\ | \\ CH_2OH \end{array} \longrightarrow \begin{array}{l} COOH \\ | \\ COOH \end{array}$$

In other words, we have here a striking example of a definite line of oxidative action, involving an attack upon a distinct group of atoms, with a complete ignoring of other groups which *a priori* are seemingly just as easy of oxidation. How can this selective action be accounted for other than by the assumption that we are dealing with specific oxidases which have an affinity as it were for distinct groups or radicals, reinforced by an influence exerted by the stereochemical configuration of the molecule acted upon. In any event, these results are a striking refutation of the view that animal oxidation follows ordinary chemical and physical laws.

This statement may be reinforced by reference to the results obtained by Mayer* in his experiments with glycol aldehyde. This substance by oxidation would naturally give rise to glycollic acid, glyoxylic acid and oxalic acid, as suggested by the preceding formulæ. If, however,

* Loc. cit., p. 151.

glycol aldehyde is administered subcutaneously to rabbits, it is at once oxidized without any formation of bodies intermediate between the aldehyde and oxalic acid; glycollic acid and glyoxylic acid, which might be expected, are not found even in traces. On the other hand, dextrose is formed in considerable quantity. This formation of dextrose after injection of glycol aldehyde is not to be ascribed to increased formation of oxalic acid, for it is noticeable that excretion of the sugar commences within twenty minutes after the aldehyde is introduced and before any acid formation could have exerted much influence. More plausible is the hypothesis, suggested by Mayer, that a certain proportion of the glycol aldehyde is directly condensed in the organism to dextrose, the oxidative power of the organism being inhibited in some degree by the toxic action of the aldehyde, and thus permitting a portion at least of the so-formed dextrose to escape combustion and thus appear in the urine. So here again we find evidence of specific oxidative action in the organism, due no doubt to a specific aldehydase, combined with a process of condensation, both of which are very suggestive in connection with our understanding of the processes involved in intermediary carbohydrate metabolism.

The formation of oxalic acid in the body, just referred to in connection with the oxidation of glycol derivatives, is worthy of further consideration in connection with carbohydrate metabolism. The work of Mohr and Salomon* has shown that the body of man normally produces a small amount of oxalic acid which is excreted in the urine. With an oxalate-free diet, the daily urine contains 2–6 milligrams of the acid, increased somewhat by certain foods such as gelatin. Practically, all the tissues of the body contain small amounts of oxalate; muscle of man, for example,

*Untersuchungen zur Phyiologie und Pathologie der Oxalsäurebildung und-Ausscheidung beim Menschen. Deutsch. Archiv. f. klin. Medicin, Band 70, p. 486.

showing 6.5 milligrams of oxalic acid per kilo of tissue.* According to Salkowski, the spleen, liver and muscle by autolysis in the presence of uric acid form a small amount of oxalic acid. More pertinent, however, are the observations of Hildebrant † who found on giving dextrose to rabbits fed on oats (which contain lime salts) that there was a very great increase in the production of oxalic acid, the acid appearing in the urine as calcium oxalate. In other words, under some conditions at least, oxalic acid may be considered as an oxidation product of dextrose.

In this connection, it is to be remembered that many observations ‡ tend to show that in the incomplete oxidation of sugar in the body, glycuronic acid is a conspicuous product. Thus, in diabetes mellitus there is frequently, if not most generally, an increased excretion of glycuronic acid; a condition which often persists even when in consequence of a strict anti-diabetic diet, sugar excretion has entirely disappeared. Moreover, there are many diseases in which sugar rarely appears in the urine where glycuronic acid is present in increased quantity. Thus, as stated by Mayer, there are various acute febrile conditions in which increased excretion of glycuronic acid is found in harmony with the fact that in these diseases the power of assimilation for dextrose is inhibited somewhat, coupled with diminished power of oxidation of sugar, thereby resulting in a portion of the dextrose being oxidized only to glycuronic acid, while the conditions do not admit of any excretion of unchanged sugar. Further, it is to be noted that when a soluble salt of glycuronic acid is given to rabbits there results a marked increase in the output of oxalic acid, proportional to the amount of glycuronate fed. In diabetes, oxaluria is occa-

* Cipollina: Ueber die Oxalsäure im Organismus. Berliner klin. Wochenschrift, 1901, p. 544.

† Uber eine experimentelle Stoffwechselabnormität. Zeitschrift f. physiologische Chemie, Band 35, p. 141.

‡ See especially Paul Mayer: Uber unvolkommene Zuckeroxydation im Organismus. Deutsche med. Wochenschrift, 1901, p. 243 and p. 262.

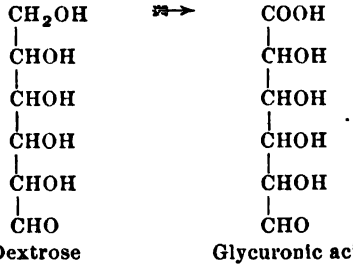
sionally met with, in harmony with a preceding statement that oxalic acid is liable to result from the incomplete combustion of dextrose. The well known antagonism between glycosuria and oxaluria frequently observed is due to the fact that in severe cases of diabetes the greater portion of the sugar present is not oxidized at all, while with improvement in the assimilation of sugar a portion is oxidized, not, however, to carbon dioxide and water, but simply to oxalic acid. From these statements, the inference may be drawn that both glycuronic and oxalic acids are products of the incomplete oxidation of sugar, and further, that in some instances at least glycuronic acid is an antecedent or intermediary body in the production of oxalic acid.

Glycuronic acid is unquestionably an important body in the intermediary metabolism of carbohydrates. It is much more widely distributed and much more common than has generally been supposed. It does not occur in the free state in the animal body, but exists conjugated with various aromatic substances, notably with phenol, indoxyl, etc., these conjugated forms being normally present in the urine. The introduction of camphor or chloral hydrate (with which glycuronic acid combines) into the system is followed by a marked increase in the excretion of glycuronic compounds in the urine, thus implying the formation in the body of much more glycuronic acid than is indicated ordinarily by the content of this substance in the urine.

As to the origin of glycuronic acid, there is abundant experimental evidence, in harmony with what has previously been stated, that in rabbits at least the acid can be formed from dextrose. Further, there is likewise experimental evidence tending to show that in all probability glycuronic acid can also originate from proteid, *i. e.*, from the carbohydrate group of the proteid molecule.* The transfor-

* See Paul Mayer: Neue Untersuchungen über die Glukuronsäure. Biochemisches Centralblatt, Band 1, p. 377.

mation which dextrose undergoes when oxidized to glycuronic acid is very slight, and consists simply in the conversion of the primary alcohol group of the sugar into the carboxyl group of the acid, leaving the aldehyde group of the sugar intact, as indicated by the following formulæ:

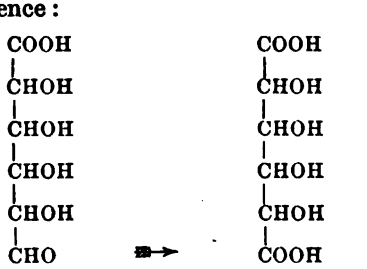


Dextrose Glycuronic acid

A priori, it might be expected that in the foregoing oxidation the very labile aldehyde groupe would be the one to undergo change with formation of the monobasic gluconic acid, but the facts available do not support this view and the conclusion may be accepted as affording another evidence of the specific action of tissue oxidation, in which without doubt intra-cellular enzymes are the active agents. The evidence at hand, both experimental and clinical, renders quite conclusive the view that glycuronic acid appears in the tissues and in the excretions as the result of an incomplete oxidation of dextrose (both that which is derived from carbohydrate and that which has its origin in the breaking down of the proteid molecule), and consequently the amount of this acid present may be taken as a measure of the extent to which the sugar is escaping oxidation or combustion. Further, it is to be understood that glycuronic acid is a normal intermediary product of carbohydrate metabolism, representing perhaps the first step in the breaking down of sugar, which under ordinary or normal conditions progresses to further stages with complete or nearly complete disappearance of the intermediary glycuronic acid, except in those cases where further oxida-

tion is wholly or in part inhibited. In the latter case, the amount of glycuronic acid in the circulation is greatly increased and so likewise the various aromatic substances, which under ordinary circumstances are combined with it in small degree, are conjugated in larger quantities, thereby leading to an increased excretion of phenol, indoxyl and other glycuronates, with a corresponding decrease in the excretion of ethereal sulphates.

If glycuronic acid represents the first stage in the intermediary metabolism of sugar, then the dibasic saccharic acid might well be suspected as the second product in a progressive oxidation, where the aldehyde group of glycuronic acid is replaced by the carboxyl group as indicated in the following formulæ, a view which is supported by experimental evidence:



Glycuronic acid — Saccharic acid

Comparison of the structural formula of saccharic acid with that of oxalic acid $\begin{matrix}\text{COOH}\\|\\\text{COOH}\end{matrix}$ shows how easily by one or more changes the latter acid may be formed by progressive oxidation, in harmony with the fact already mentioned that administration of glycuronic acid is followed by an increased excretion of oxalic acid, accompanied by accumulation of oxalic acid in the liver. The liver is unquestionably one of the places where glycuronic acid is oxidized to oxalic acid, since it is easy to demonstrate by digestion experiments with comminuted liver tissue the transformation of the former acid to oxalic acid; an observation of

obvious importance in intermediary metabolism. Finally, it may be mentioned that there is some ground for the belief that a part of the dextrose in the body is normally oxidized without first passing through the intermediary stage of glycuronic acid. How large this fraction may be cannot be said, but in any event the glycuronic acid that is formed is further oxidized with subsequent formation of a variable amount of oxalic acid.

As we study these various intermediary steps and stages in carbohydrate metabolism, we cannot avoid being impressed more and more fully with the general principle that the appearance of the so-called abnormal products of carbohydrate degradation, like sugar itself, is due not so much to perverted metabolic action as to inhibition of one or more of the normal oxidative processes by which, under normal conditions, these various intermediary products are further oxidized and destroyed. Incomplete or improper burning of sugar is responsible for the presence of these symbols of disturbed nutrition. Under ordinary conditions they are quickly oxidized from stage to stage, through the agency of specific oxidases or other ferments, until the final end-products are reached. Disturbance of function may therefore be ascribed in ultimate analysis to lack of the specific enzyme, or to lack of suitable conditions for the proper action of the enzyme. The burning of sugar in the body is as much dependent upon the progressive action of specific oxidizing enzymes as the hydrolysis of carbohydrates in general is dependent upon the action of amylolytic enzymes. This principle is well illustrated by some experiments recently carried on in our laboratory by Mr. P. H. Mitchell, in which it was found, for example, that soluble erythrodextrin when injected subcutaneously or intraperitoneally was thrown out through the urine in large amount as achroodextrin; *i. e.*, essentially unchanged. Though readily oxidizable, by this method of introduction the dex-

trin escaped contact with the enzymes which under other conditions would have quickly oxidized and destroyed it. Similarly, Mr. Mitchell found that the carbohydrate inulin introduced in like manner could be recovered in the urine to the extent of 70 per cent. wholly unchanged. Dr. Paul Mayer has reported a similar experience with glycogen, recovering 50 per cent. of the carbohydrate essentially unchanged in the urine after subcutaneous injection. In other words, the specific enzymes, the intra-cellular ferments of the tissues, as well as the amylolytic enzymes of the digestive juices, must have proper opportunity for action if the successive steps of carbohydrate metabolism or oxidation are to be successfully carried out.

Again, in many forms of experimental diabetes such, for example, as we have been in the habit of speaking of as due to "insults" offered to the pancreas, where such substances as piperidin, potassium cyanide, morphin, coniin, nicotin, curare, ether, chloroform, etc., produce marked hyperglycæmia and glycosuria, the explanation now offered* is that these drugs do not have any specific action upon any particular gland, like the pancreas, but that they exert more or less of an influence upon the respiratory centre, producing dyspnœa. Experiments conducted in our laboratory by Dr. Underhill have shown, for example, that while piperidin painted on the pancreas causes marked hyperglycæmia and consequent glycosuria, the same result follows when the piperidin is painted on the spleen, introduced intraperitoneally, or injected directly into the blood. Further, the drug fails to produce these results when oxygen is administered. In other words, as stated by Dr. Underhill, experimental diabetes of this sort is due simply to diminished oxidation of carbohydrate material, with the consequent accumulation of sugar in the blood, and elimination by the

* Underhill: Certain Aspects of Experimental Diabetes. American Journal of Physiology, Vol. 13, p. xxxvi.

kidneys. Dyspnœa is always prone to call forth marked hyperglycæmia and glycosuria, whether induced by drugs or otherwise. Lastly, may we not query whether the lack of oxidation of sugar and the consequent glycosuria in these cases is not due to conditions unfavorable or inhibitory to the action of the normal oxidases or other ferments which ordinarily are effective in destroying sugar?

Another chapter in intermediary metabolism well worthy of consideration is that which relates to the amino-acids; but time will not permit of more than a passing reference to these interesting substances, although many of them are formed as products of proteid decomposition within the body, and play important parts in intermediary metabolism. Some of them, however, are connected with peculiar anomalies of proteid katabolism, and I may therefore be pardoned if I refer to one or two, since they are associated with conditions of the body in which certain metabolic functions are strangely perturbed. Cystin (and the related body, cystein) is well known as a primary crystalline decomposition product of proteids and is characterized by containing the sulphur of the proteid molecule from which it is derived. In that somewhat rare disease, known as cystinuria, cystin appears in the urine, sometimes in fairly large amounts. Normally, the cystin which must be split off from every sulphur-containing proteid during the process of katabolism is oxidized or destroyed by the processes of intermediary katabolism and disappears. But in cystinuria, the body loses the power of burning or oxidizing this substance, and hence it is excreted unchanged. Now, it is claimed that there are two, isomeric, forms of cystein: one, known as protein-cystein, because of its production from horn and other proteids, and having the formula: $CH_2 . SH - CH . NH_2 . COOH$, *i. e.*, α-amino-β-thio-propionic acid; while the other, known as calculus-cystein, because obtained from urinary calculi, has the formula:

$CH_2 . NH_2 - CH . SH - COOH$, *i. e.*, *α*–thio–*β*–amino-propionic acid. If the so-called protein-cystein is fed to a person afflicted with cystinuria the substance is excreted unchanged, simply increasing the amount contained in the patient's urine; but if the isomeric form, calculus-cystein, is fed it at once disappears, the amount of sulphate in the urine being correspondingly increased, just as happens with both forms when introduced into the system of the healthy individual.* In other words, under the conditions prevailing in cystinuria, the body has lost the power of burning the one form of the amino-acid, *i. e.*, the *α*-amino acid. Indeed, in this condition of disease, intermediary metabolism is so perverted that all *α*-monamino acids whenever introduced into the system are excreted unchanged, the body having apparently lost all power of burning them. Thus, the *α*-monamino acids, tyrosin, leucin, aspartic acid, etc., which are completely oxidized in the healthy organism to carbon dioxide and ammonia, *i. e.*, as urea, are excreted almost quantitatively when fed in cystinuria. We may well pause here to emphasize the fine discrimination which the body shows in its treatment of these two isomeric forms of cystein (in cystinuria). The two substances are essentially alike, except in the relative position in the molecule of the amino (NH_2) group, yet the one is easily oxidized, while the other passes through the body absolutely unchanged. Moreover, it is evident that in cystinuria this inhibition of the power to burn the *α*-amino acid is a general one, preventing any *α*-amino acid from undergoing change in the body.

Further, in cystinuria, diamino-acids, such as arginin and lysin, behave in a peculiar manner. Unlike the monamino-acids, they undergo change, but it results simply in a slight transformation and not in a complete or

* See Loewy and Neuberg: Ueber Cystinurie. Zeitschrift f. physiologische Chemie, Band 43, p. 338.

profound oxidation. Thus, when arginin is fed to a person with cystinuria it is converted into the diamine putrescine, viz., tetramethylene-diamine:

$$NH_2.C(NH) - NH.\ CH_2 - (CH_2)_2 - CH.\ NH_2 - COOH \rightarrow$$

Arginin

$$NH_2 - CH_2 - (CH_2)_2 - CH_2\ NH_2$$

Putrescine

In a similar way the diamino-acid lysin is transformed into the diamine cadaverine, viz., pentamethylene-diamine:

$$CH_2NH_2 - (CH_2)_3 - CH.\ NH_2.\ COOH \rightarrow$$

Lysin

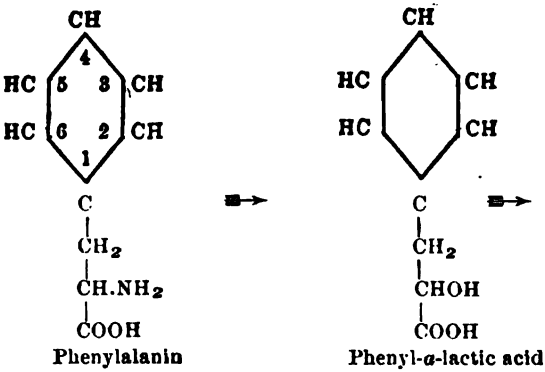
$$CH_2.\ NH_2 - (CH_2)_3 - CH_2.\ NH_2$$

Cadaverine

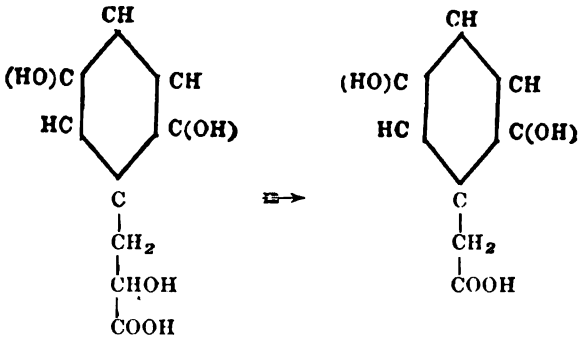
With these facts before us as illustrations, it is evident that in such a disease as cystinuria many changes of metabolism may occur, by which intermediate products ordinarily undergoing progressive oxidation are prevented from suffering further change, and are hence cast out of the body unaltered. Their presence in undue quantities, however, is not without possible physiological significance. For example, it needs very little imagination to see how, when the body has lost in greater or less measure the power to burn organic acids, a condition of acidosis may be produced, and how this condition may in turn influence the production of ammonia in the body from the breaking down of proteid in an attempt on the part of the latter to neutralize or overcome the influence of the accumulating acid. But we have not time to discuss this theme, however important or suggestive it way be. Rather let me bring to your attention another anomaly of intermediary metabolism, as seen in alkaptonuria, a condition in which the urine shows the presence of the peculiar diphenol acids known as homogentisic and uroleucic acids. It has long been known that in this disease a rich proteid diet tends to increase the

output of these acids, from which the inference has naturally been drawn that some cleavage product of the proteids must be their antecedent. The presence of the aromatic group suggests tyrosin as one of the antecedents, and experiment has shown that the ingestion of tyrosin by persons having alkaptonuria is followed by an increased excretion of homogentisic acid. The same observation has been made with phenylalanin, from which we may conclude that both of these aromatic substances, derivatives of proteid matter, are normal antecedents of homogentisic acid. In other words, the characteristic acids found in the urine in alkaptonuria have their origin in the aromatic complex of the proteid molecule through the stage of the two aromatic substances mentioned above. As stated by Falta,* it is quite apparent that in alkaptonuria there is a profound disturbance in the breaking down of the aromatic amino-acids which result from proteid katabolism, as a result of which uroleucic or homogentisic acid (or both) appear in the urine. Under normal conditions, these two acids after suffering deamidization are completely oxidized to carbon dioxide and water. Thus, in the normal individual, homogentisic acid introduced into the alimentary tract completely disappears, *i. e.*, it is entirely oxidized to simpler products, but when fed to a person afflicted with alkaptonuria it appears quantitatively in the urine. In other words, in alkaptonuria the body has entirely lost the power of splitting off the benzol ring from the intermediary proteid product homogentisic acid. On the basis of experimental evidence, it is probable that the progressive breaking down of the aromatic amino-acids derived from proteid katabolism, after having reached the tyrosin or phenylalanin stage, is as follows:

* Die Alkaptonurie. Biochemisches Centralblatt, Band 3, p. 173.



Phenylalanin — Phenyl-α-lactic acid



Uroleucic acid — Homogentisic acid

Especially interesting and suggestive in this schematic representation of the intermediary changes from phenylalanin to homogentisic acid is the formation of phenyl-α-lactic acid. The splitting off of nitrogen from the alanin, *i. e.*, its deamidization, takes place at this stage with the resultant formation of a non-nitrogenous acid, a complex of the aromatic group and α-lactic acid. We thus see opened up, as stated by Falta, a new point of view regarding the formation of sugar from proteid. Between lactic acid and dextrose there are many obvious physiological connections and it is quite conceivable, to say the least, that a portion of the lactic acid originating in this manner may in the liver

be transformed into sugar or glycogen. In alkaptonuria, however, while deamidization of alanin may occur as normally, oxidation and other changes stop at the uroleucic or homogentisic acid stage, the body cells having lost the power of splitting off or burning the benzol ring; an anomaly in intermediary metabolism without doubt due to the absence or inhibition of the specific intra-cellular enzyme which normally accomplishes this transformation.

And so we see on every hand in studying the many and varied processes of intermediary metabolism, of which the foregoing are to be taken merely as illustrations, the significant part played by intra-cellular enzymes in bringing about the normal and abnormal changes characteristic of health and disease.* The processes which we have heretofore considered as due to the peculiar vital properties of the tissue cells, and which were supposed to be entirely dependent upon their morphological and functional integrity, we now see are due — certainly in large measure, if not wholly — to a great variety of enzymes which can be separated from the cell substance and which retain their activity even when free from the influence of the living protoplasm. Further, we find ample evidence that the varied processes of katabolism are the result of orderly chemical changes, of a progressive character, in which cleavage, hydration, reduction, oxidation, deamidization, etc., alternate with each other under the influence of specific enzymes, where chemical constitution and stereochemical configuration of the various molecules are determining factors in the changes produced. It is therefore evident that our understanding of intermediary metabolism will depend in large measure upon the intelligent application of the principles and methods of physiological chemistry in our study of these important and far-reaching problems.

* See A. Oswald: Die Bedeutung der intracellulären Enzyme in der Pathologie. Biochemisches Centralblatt, Band 3, p. 365.

ARTICLE III.

WHAT THE SUBURBAN SURGEON IS DOING IN THE ABDOMEN, AND HOW HE DOES IT.

By CHARLES E. DURANT, M.D.
OF HAVERHILL.

READ JUNE 13, 1905.

WHAT THE SUBURBAN SURGEON IS DOING IN THE ABDOMEN, AND HOW HE DOES IT.

In presenting this series of abdominal sections for your consideration, it is not my purpose to attempt a recital of unique experiences or new methods, but to give an account of the manner in which I have dealt with the various conditions herein recorded, as they have occurred in suburban practice, together with a statement of the results obtained.

Suburban hospitals have multiplied with great rapidity in recent years, and, in consequence, much major operating is being done by local men, whose methods and whose results, aside from the occasional report of a case of perhaps more than ordinary interest, are to a great extent unknown. This is not as it should be, for there are many problems relating to this class of surgery which could be much more easily disposed of if all the facts were available. With this idea in mind, I shall offer a report of my personal experience, in the hope that it may contribute in some measure to a knowledge of what the suburban surgeon is doing in the abdomen, and how he does it.

I have divided the list into groups, as follows: I. Appendicitis; II. Pelvic Conditions; III. Disease of the Gall-Bladder and Bile Ducts; IV. Hernia; V. Miscellaneous conditions not easily classified.

Group I.—Appendicitis.

Appendicitis: 125 cases, 97 acute and 28 in the interval. Of the acute cases 14 died, a mortality of 14.3%; in the

interval cases there were no deaths. A somewhat critical study of the cause of death in the fatal cases will serve to explain this high death rate.

In seven cases general peritonitis was present at the time of operation. These cases had either not been recognized as appendicitis or operation had been refused, or delayed, until the patient's condition became desperate. In the face of such odds, one is always tempted to do nothing, for if the patient dies, as he usually does, the surgeon gets no credit, and may get much blame. On the other hand, we know that after a rapid removal of the focus of infection, a certain small percentage of these cases recovers. It is clearly our duty, then, it seems to me, to advise operation, in disregard of all other considerations, for it offers the patient his only chance.

When the general practitioner becomes convinced that appendicitis is a surgical disease, and that early operation gives the best results, the surgeon will rarely be confronted with this condition. Many practitioners, however, especially among the older men, are still extremely conservative, and one often hears in the District Societies and in private conversation, rather forcible language against what is called too much operating in appendicitis, from men of local consequence and large clientele. The demonstration of morbid conditions *in vivo* does not always impress them, and I fear that the teaching of Ochsner, while perhaps of advantage in certain cases and under certain conditions, will have an influence to delay progress in this direction.

Two of the earlier cases died of late sepsis, the result of post-cecal collections of pus. I should not, at the present time, expect a fatality from this cause alone. One patient, a little girl of three years, died in convulsions 18 hours after operation, in what appeared at the time to be a favorable case.

One death occurred from hemorrhage, the result of gan-

grenous involvement of the iliac vein. A little girl of ten, a patient of Dr. Peirce of West Newbury, was operated upon the fifth day of the attack. The appendix was found behind the cecum, markedly gangrenous. Everything went well for a week, when the attending physician, upon removing the gauze drain, observed a considerable gush of venous blood from the wound. It was so profuse that firm packing was necessary to control it. I saw the child soon afterward, and made a hasty exploration, but not being able to definitely locate the source of the trouble, I repacked the wound with gauze. The hemorrhage, however, continued, and the patient died in about 36 hours. J. Ryerson Fowler reports a similar case in his monograph.

Three patients died of peritonitis on the fourth, sixth and seventh day, respectively. In dealing with such cases, I think one should always consider the advisability of enterostomy, as suggested by Greenough, although Richardson states distinctly that he has never seen any benefit come from it.

One patient, a lad of nine, whose abdominal condition was entirely satisfactory, died of acute pneumonia on the fifth day.

Aside from complications already cited, there were two cases of post-operative obstruction, requiring a second operation,—one early, upon the eighth day, and one late, after four months. In the first case a parietal adhesion had caused sharp angulation of the intestine at the point of attachment, resulting in complete occlusion; in the other a narrow band was found in the right iliac fossa, completely encircling the ilieum; both recovered.

Three patients were operated upon during pregnancy: two in acute attacks, one at seven months, the other at three months, and one in the interval at three months; all recovered. In the first case the patient was prematurely delivered on the eighth day; in the other two pregnancy

was not interrupted. In one case right tubo-ovarian abscess and fibro-myoma of the uterus were found as complications. This patient recovered after removal of the abscess and appendix, and six months later also recovered from hysterectomy for removal of the fibroid.

Excluding the six cases of general peritonitis, all of which were *in extremis* and almost certain to die under any treatment, the mortality was 8.2%. I mention the two sets of figures for the purpose of emphasizing by comparison what seems to me to be the importance of early operation; and this naturally leads to a brief consideration of that question,—the only one, in fact, aside from technique, to which I shall give any attention.

In 1902, Meyer and Elsberg published papers on "What can be diagnosticated in appendicitis," and while it seemed reasonably certain that the position of the appendix could often be determined, they concluded that nothing definite as to the pathological condition could be predicated from signs and symptoms. My own experience has impressed me with the absolute soundness of this doctrine.

For example, I have found a completely gangrenous appendix in a case where the pulse and temperature were normal and where no rigidity and only slight tenderness were present; I have found the inflammatory process limited to the mucous membrane, with only slight injection of the peritoneum of the appendix, in a case where a history of sudden severe and continuous pain, marked tenderness and rigidity, with a pulse of 130 and a temperature of 104°, would clearly suggest something worse. And so one might go on with an almost endless recital of similar experiences.

Furthermore, if my observations are correct, there is no way by which the probable course of the disease may be predicted in a given case. It is true that many attacks of appendicitis subside, even when the severity of the symptoms would not warrant the probability of such a result.

On the other hand, it is not too much to say that every surgeon sees cases which appear mild for a day or two, then suddenly grow worse, and demand operation and very likely drainage,—a thing to be avoided if possible, both on account of present as well as future disadvantage.

In deciding this question, then, one is confronted with certain facts: first, the entire uncertainty of the nature of the process present; second, no reliable information as to the course which the disease is likely to pursue; third, the knowledge that in waiting one takes the chance of being too late or of subjecting his patient to drainage. This, it seems to me, is a fair and plain statement of the question. If we accept the premises, the conclusion is inevitable.

Technique:

In preparing a patient for operation in acute cases, an enema is first given. The field is shaved, scrubbed in soap and water for five minutes, using sponges instead of a brush, and then washed with 70% alcohol. Especial attention is given to the umbilicus, which is finally painted with tincture of iodine. The operator's hands are thoroughly scrubbed with soap and water for five minutes, using a stiff brush; they are then immersed in 70% alcohol for two minutes. Gloves are always worn. I have varied the method in some cases by washing the field in solution of chlorinated soda 1-10 before the alcohol is applied. I have also occasionally used solution of biniodide of mercury, 1-500 in alcohol, as suggested by Treves. The instruments, including needles, are boiled for ten minutes. Scalpels, after being thoroughly washed, are immersed in either 5% solution of carbolic acid or 70% alcohol, preferably the latter.

My incision is vertical through the outer border of the right rectus, beginning about two inches below the level of the umbilicus. This, as Routier says, always leads to the cecum, and is less likely to be followed by hernia if drainage

is used. It also has the advantage of being easily enlarged if pelvic complications require interference. It is sometimes bloody if the deep epigastric vessels are encountered, but this is of small moment.

Immediately upon opening the peritoneum, it is my practice to dispose of gauze strips in such a manner as to fully protect the general cavity, before attempting to locate or remove the appendix. If turbid serum or pus is present, it is carefully wiped away with sponges.

I usually remove the appendix by the so-called German method, which consists in clamping the free border of its mesentery at two points and cutting between them with scissors. A few more snips through the base of this mesentery, close to the appendix, brings one to the cecal junction. While making these cuts with scissors, it is necessary for the assistant to catch two or three bleeding points on the mesentery and one or two on the appendix. Those on the mesenteric stump are tied at once with plain catgut, but the clamps are left on the appendix without tying. A No. 2 or 3 chromic gut ligature is tied around the appendix close to the cecum.

The appendix is now ablated with scissors or the cautery. If scissors are used, they are discarded from the kit, and the stump is touched with pure carbolic and absolute alcohol. The artery forceps used for this purpose are also discarded. It is often much easier to remove the appendix base first in cases where the tip is firmly adherent, or where its location is not at once evident, on account of twists or angulations.

The question of whether or not drainage should be used in certain acute cases is often difficult to decide. Where only turbid serum is present, or where there is but little pus, I have of late been in the habit of closing the abdomen, after carefully wiping away the serum or pus with gauze sponges. Such cases cause a certain amount of anxiety for a day or two, but I believe to a considerable extent in

the teaching of Morris, that we may place great confidence in the absorptive and phagocytic power of the peritoneum after the source of infection is out of the way.

In drainage cases, I invariably use the cigarette, placing a small wick down to the stump, or behind the cecum, and sometimes one into the pelvis. The abdomen is generally closed down to the drains with mass silkworm gut suture. Recently I have been making a much shorter incision than formerly, and closing it with tier sutures, leaving but a small opening for the wick. This procedure seems worth while, for by protecting the lower end of the suture line with rubber tissue, one often gets first intention, and with it the advantages of a wound closed in layers, minus a small cicatrix at the lower end. If the wound breaks down and heals throughout by granulation, the shorter it is the better.

In chronic cases I employ the incision of Lennander at the outer border of the right rectus, with retraction of the muscle inwards. The patient is placed in moderate Trendelenberg position and the abdominal wall is lifted away from the intestines with retractors. This manœuvre enables one to see the cecum, and where there are no adhesions, the caput coli and appendix can be easily lifted out of the abdomen, with practically no evisceration and very little handling. The appendix is removed as I have already described.

I have found the method suggested by Kelly to be of the greatest service where the appendix is surrounded by a dense mass of exudate. The base is first detached; a circular incision is then made through the peritoneal and longitudinal muscular coats. The remaining portion of the appendix is seized with forceps and stripped out of its bed without difficulty and with no hemorrhage.

It has not been my custom to bury the stump by purse string or other procedure, although I realize that most sur-

geons consider it good practice. Recently, however, Lilienthal and Seelig have shown quite conclusively, it seems to me, that this procedure is not only unnecessary, but may be really dangerous.

In closing I use the layer suture, consisting of continuous plain catgut in the peritoneum and posterior sheath of the rectus, two or three interrupted plain catgut sutures under light tension in the muscle, chromic gut or kangaroo tendon mattress sutures in the fascia, two or three silkworm gut tension sutures with a double loop in the fat, and a continuous lock-stitch horsehair to approximate the skin.

Post-operative Treatment:

Nothing is given by mouth for six hours, but if thirst is great, enemata of salt solution are used. At the end of this time the patient is allowed frequent sips of hot water, and, if well borne, for two or three hours small amounts of milk and lime water, or milk and Vichy are given, and gradually increased. Morphine is generally forbidden and very rarely found necessary. Pain after operation is usually dependent upon the degree of distention present, and for the relief of this condition I have found injections of milk of asafœtida to be of great service. If it is necessary to do anything further for pain, I use fifteen to twenty grains of aspirin by rectum. I consider it desirable to have the bowels move as soon as possible after operation, especially in pus cases, and for this purpose I use fractional doses of calomel up to one or two grains, to be followed in two hours after the last dose by a high compound enema of Epsom salts, turpentine and oil, or glycerine.

Group II. — Pelvis.

There were eighty-nine operations upon the pelvic organs for various conditions, including twenty-five cases of pyosalpinx; thirty-one cases in which section was done for ovarian disease or displacement, and prolapsus or retrover-

sion of the uterus, with or without adhesions; fourteen cases of uterine fibroids, nine of which were treated by hysterectomy and five by myomectomy; ten cases of ovarian cysts, four of which were dermoids; two cases of contracted pelvis and one case of placenta previa, in which Cæsarian section was done; five cases of extra uterine pregnancy with intra-peritoneal rupture; one case of cancer of the cervix, treated by total hysterectomy.

In this series there were five deaths,—three following operations for pyosalpinx, one in a case of large serous cyst of the ovary with papillomatous degeneration and ascites, in a feeble patient, and one from mechanical obstruction by a band, following hysterectomy. In the last case death could have been prevented, in all probability, by a second operation, for the autopsy showed a band across the descending colon, easily accessible. The site of the operation was clean.

Of the twenty-five cases of pyosalpinx, fifteen were single and ten double. There were three cases in which rupture had occurred into the general peritoneal cavity, with one death. In the other two fatal cases there were large collections of pus in both tubes; the walls were greatly thickened and the whole mass so densely adherent that lines of cleavage could not be developed and enucleation was impossible. Both were septic; one died from shock a few hours after operation, the other lived a week and died from sepsis. There were no post-operative complications of note, except in one case, where a second laparotomy was necessary to relieve obstruction caused by a parietal adhesion, and three cases of left-sided femoral phlebitis, following hysterectomy for fibroid, ovariotomy, and operation for bilateral non-suppurative tubo-ovarian inflammation with extensive adhesions, respectively.

The appendix was removed whenever found diseased on gross appearance or adherent, and, as a rule, the uterus

was suspended. Drainage was used in all but two of these cases.

In one of the non-suppurative cases operated on for ovarian and uterine displacement, a dermoid the size of an English walnut was found connected with each tube near the fimbriated extremity. Of four cases of dermoid cyst of the ovaries, one proved to have a twisted pedicle, and the diagnosis of appendicitis which was made before the patient entered the hospital seemed fully warranted. One nullipara, 24 years old, suffered from marked uterine prolapse, and was treated by ventro fixation. In the fibroid cases there was nothing of interest, except that one patient developed mental confusion, with ideas of persecution, on the third day after hysterectomy. She had worried a great deal for a year about her condition, and at the time of operation was much depressed. She recovered completely in a week, the post-operative course of the case otherwise being perfectly normal. The extra-uterine cases were all typical.

Technique:

In operating for pus tube, the patient, after being curetted, is placed in moderate Trendelenberg position, and the abdomen opened in the median line. The intestines are pushed back out of the pelvis and carefully protected by gauze packing. If the omentum is adherent, which is often the case, it is gently separated with the fingers or cut between two ligatures.

The key to the operation for pyosalpinx is the line of cleavage between the tumor and surrounding structures; and having found this, it is my custom to shell out the mass, using the ends of two or three fingers for this purpose. One should be especially careful when separating the sac from the rectum. If rupture occurs during the manipulation, the pus is quickly taken up with sweeps. After all adhesions are separated, the upper edge of the broad liga-

ment, including the ovarian artery, is tied on either side of the tumor with kangaroo tendon. The tumor is now ablated by a wedge-shaped cut through the broad ligament, the edges of which are sewed together with a continuous lock-stitch of chromic gut. The uterus is usually suspended with two kangaroo tendon sutures through the anterior wall. A cigarette drain, which in my practice has grown much smaller of late, unless there is considerable oozing, is placed in the pelvis, and the abdomen partly closed in layers.

In pelvic conditions characterized by prolapsed and tender ovaries, and posterior displacements, with or without adhesions, not accompanied by other demonstrable lesion, I have learned by experience that one should be very cautious about deciding to operate, and when the decision is made, one should be still more cautious about what is done and how much is promised, for it must not be forgotten that many neurasthenic women have pelvic symptoms, such as painful and tender ovaries, painful menstruation, sacral ache, and perhaps retro-version, who will not be benefited by a surgical operation, even though it be most conservative. If, after careful consideration, one is satisfied that the pelvic condition is primarily the cause of the trouble, or is contributory in large measure to its continuance, he should operate.

If the ovary is prolapsed into Douglas's pouch and fixed there by adhesions, it is freed, and after being resected, if distinctly cystic, it is sutured to the side of the uterus just below the tubal junction with the chromic gut. In resecting, a wedge-shaped piece is removed with the knife and the line of section closed with plain catgut. Before concluding, however, that an ovary is cystic, one should make sure, as Craig suggests, that he is not dealing with normal Graafian follicles nearing the time of rupture. The uterus is likewise freed, if adherent, and suspended by two

kangaroo tendon sutures through the anterior wall and through the edges of the peritoneum.

Sufficient evidence has now been accumulated to establish the value of Kelly's operation in the treatment of retropositions, but that it sometimes fails of its purpose mechanically there can be no question. As an illustration of this fact, I will cite the case of a patient upon whom I did suspension in 1901, and unwisely closed the small incision with a mass suture of silk-worm gut. She presented herself three years later with a ventral hernia, for which I did a second operation. Upon opening the abdomen, the uterus was found in marked retro-position, my peritoneal ligament being stretched out into a mere string of tissue.

Prolapsus in women past the menopause is treated by ventro fixation with two silk sutures, which are placed in the anterior wall of the uterus and include the fascial layer of the abdominal wall above which they are tied. It is usually necessary to supplement this with plastic work on the anterior vaginal wall and perineum.

In supra vaginal hysterectomy for fibroids, the broad ligaments, including the ovaries if diseased, are tied with kangaroo tendon on the pelvic side and clamped on the uterine side nearly to the level of the uterine arteries. A transverse incision is then made across the front of the uterus, just above the attachment of the bladder, and the peritoneum dissected back for perhaps an inch. A corresponding incision is made posteriorly and the peritoneum dissected back in the same way. The uterine arteries are then tied with a ligature, carried beneath them by the Cleveland needle, bearing in mind while doing this the close proximity of the ureters. The uterus is ablated with scissors in an antero-posterior wedge-shaped section. The cervical canal is cauterized with pure carbolic acid or the actual cautery, and closed with chronic gut. The peritoneal surfaces of the broad ligaments and over the stump are brought

together with a continuous lock-stitch of chromic gut or small kangaroo tendon. There were five myomectomies in this list in cases where the tumors were single and pedunculated. A wedge-shaped section was removed from the uterine wall to include the proximal extremity of the pedicle; the wound was closed with chromic gut.

Ruptured extra-uterine pregnancy, if not promptly recognized, is one of the greatest catastrophes that can befall a woman, and I have repeatedly warned my confreres to be on the lookout for this condition. The abdomen should be opened as quickly as possible, and after clearing the field of blood clot, the free border of the broad ligament is clamped on either side of the ruptured sac to control the hemorrhage. A partial salpingectomy is then done in the ordinary way. I make no effort to remove the extravasation,— it takes too much time. It is quite necessary to have help enough on hand to infuse when the operation is begun, and equally necessary to use all possible means to combat post-operative shock. In two of the five cases in this series, all of which recovered, I am satisfied that intravenous infusion turned the scale.

Among three Cæsarian sections I will only speak of one,— a case of placenta previa. As to whether or not the operation is ever indicated in this condition, there is a considerable difference of opinion, and I shall not discuss it, except to say that in this case, after careful consideration (Drs. Croston, I. J. Clarke and O'Donnell being in consultation), it seemed to offer by far the best chance for the child, and not greatly increased danger to the mother.

Mrs. G., æt. 30, primipara eight months pregnant, a patient of Dr. Croston of Haverhill, was taken suddenly at 5.30 A.M., with a considerable uterine hemorrhage, which shortly ceased; there were no labor pains. Placenta previa centralis was recognized,— the os was rigid and there was no dilation. At 9 P.M. she had another sharp hemorrhage, with no change in other conditions, and it was de-

cided to operate. The patient was taken to the Hale Hospital. The abdomen was opened in the median line from just above the pubes to the umbilicus. The uterus was delivered and gauze packing introduced all about it, and while the assistant tightly grasped the lower segment, an ample incision was made in the anterior wall. The child was quickly delivered, the cord being cut at once between two clamps. The placenta was attached to the lower segment of the uterus, completely covering the internal os. It was detached without difficulty and with no great amount of hemorrhage. At this point the patient was given a dram of fl. ext. ergot under the skin. Towels wet in hot salt solution were applied to the uterus, which promptly contracted. The uterine incision was then closed with three layers of chromic gut, the deepest layer not penetrating the mucous membrane. The abdomen was closed with mass silk-worm gut suture, with chromic gut in the fascia.

The other two Cæsarian sections were done in cases of pelvic deformity, the result of congenital dislocation of the hip. Both mother and child recovered in all three cases.

Group III.—Gall Bladder and Bile Ducts.

Twelve cases of disease of the gall bladder and bile ducts, including two cases of acute cholecystitis, one of which had ruptured; two cases of chronic empyema of the gall bladder, both of which had ruptured,—one into the peritoneal cavity, and the other, protected by adhesions, resulted in a secondary abscess behind the gall bladder; five cases of chronic trouble, in which stones were found limited to the gall bladder or cystic duct; one case of extensive disease of the bile tract, without gall stones; one case in which only light adhesions were found; and one case of calculous obstruction of the intraduodenal portion of the common duct. The only death in this series occurred from rupture of an empyema into the peritoneal cavity.

This list, while not large, affords an experience of some

interest in the surgery of the gall bladder and bile ducts. I will report several cases very briefly, as illustrations.

CASE I.—Female, 44, a patient of Dr. Murphy of Amesbury, was taken four or five days before I saw her with what appeared to be a typical attack of appendicitis. She gave a history of several previous attacks of a similar nature. Upon opening the abdomen, it was found to contain a considerable quantity of bile-stained fluid. The appendix was normal. The gall bladder was found to be much enlarged, livid in color, and very tense. It was adherent posteriorly in a mass of exudate of recent origin, which had evidently closed the rupture. Believing this to be the fact, I did not attempt to free the gall bladder, but made an opening in the anterior wall, and after evacuating quite a quantity of dirty brownish fluid and three large gall stones, one of which was impacted in the entrance of the cystic duct, I drained. The space between the ascending colon and the parietes, where most of the extravasated material had collected, was wiped out with sponges and the abdomen partly closed. The patient made an excellent recovery. The outcome in this case was hardly to be expected, for we are taught that the infection in acute gangrenous cholecystitis is exceedingly virulent, and that where the process goes on to perforation, a fatal result is practically certain.

To show that gall stones are not always found in the presence of other marked pathological changes in the gall bladder and bile ducts I will report the following case:

CASE II.—Female, 41, a patient of Dr. Croston of Haverhill, had been jaundiced for many weeks. She had suffered from persistent stomach disturbance, and had lost a great deal of flesh. Tenderness on deep pressure was present in the region of the gall bladder, but aside from this the physical examination was negative. We supposed the case to be one of stone in the common duct. The gall bladder was found contracted, thickened and adherent, and it contained no calculi. The foramen of Winslow was obliterated. Enlarged glands were felt in the gastro-hepatic omentum; the head of the pancreas was swollen and indurated, but no stones could be felt in the common duct. The gall bladder was opened and found to contain some bile.

Drainage by rubber tube. The jaundice began to grow less marked in about two weeks and finally wholly disappeared. The fistula closed in seven weeks, and the patient is at present perfectly well. Occlusion of the common duct in this case was unquestionably due to external pressure, caused either by the head of the pancreas, or enlarged lymph glands in the gastro-hepatic omentum, the entire process being an expression of infection in the bile tracts, in which gall stones may or may not have been a part. In any event, drainage resulted in cure.

Robert Morris has recently called attention to the importance of light adhesions between the gall bladder and adjacent viscera, in producing symptoms suggestive of gross disease of the bile tracts. He has named them gall spiders, and believes that they are the result of repeated mild attacks of acute cholecystitis. A case of this kind, which seems typical, may be cited.

Case III.—Female, 41, a very nervous patient of Dr. Young of Haverhill, had suffered for five years with attacks of severe pain in the right hypochondrium, associated with acidity, flatulence, nausea, and usually vomiting. The pain was referred to the back and right shoulder. These attacks almost invariably began late at night or in the early morning, when the stomach was empty. Recently the attacks have become more frequent and more severe. I saw her in consultation, made a diagnosis of gall stones, and advised operation. She also consulted a prominent Boston surgeon, by whom she was informed that hers was a typical case of gall-stone trouble, and that operation for this condition should be done at once.

I operated shortly afterwards, and found nothing whatever to explain her symptoms, except a few filmy adhesions between the gall bladder and pylorus. These adhesions were separated and the abdomen closed. Recovery from the operation was rapid and smooth. After the operation, the various functions of the stomach were investigated by Dr. M. P. Smithwick of Boston, and found to be normal. She has been perfectly well for eight months, except for an attack of indigestion in May, in which the nervous element seemed very prominent. This followed a period of very

hard work and great anxiety occasioned by the illness of an intimate friend. Whether or not this indicated a return of the trouble, I cannot say.

The question of how best to remove gall stones impacted in the intra-duodenal or retro-duonal portion of the common duct, has called forth some discussion, and the matter is by no means settled. This can be accounted for by the rarity of the condition, and the fact that but few cases have been reported. Two methods are in general use, viz:

Trans-duodenal choledochotomy, first performed by McBurney in 1891. In this operation the second part of the duodenum is opened. If the stone is impacted in the ampulla of Vater, the papilla is either dilated (a modification of McBurney's technique, suggested by Collins), or incised and the stone scooped out. If the impaction is higher up in the intra-duodenal or retro-duodenal part of the duct, the posterior wall of the gut is opened, the duct incised, and the stone removed. No sutures are necessary if the papilla is slit up or the intra-duodenal portion opened, but the edges of the incision in the duodenum should be sutured to those in the common duct, if the latter has to be incised after it leaves the wall of the intestine. The opening in the anterior wall of the duodenum is closed by two rows of sutures.

The other procedure, known as supra- or retro-duodenal choledochotomy, is credited by Moynihan to Haasler, and by Bryant to Berg. In this operation the duodenum is mobilized to the left, after a longitudinal incision through the peritoneum to the right of its second portion. This manœuvre, it is said, affords easy access to that part of the common duct in question. As to which of these two procedures is the more rational, I will not venture an opinion.

Thienhaus, in the Annals of Surgery for Decembor, 1902, reports twenty-nine cases of trans-duodenal choledochotomy by various operators, including one of his own, with two

deaths,— a mortality of a little less than seven per cent. He thinks that Mayo Robson's objection to this operation, based largely upon the idea that the danger of sepsis is greatly increased by opening the intestine, is not well founded, inasmuch as the published cases operated upon by the McBurney method show but a slightly higher death rate than is claimed by him as a future possibility in common duct cases, and that further evidence is necessary to settle the question. He cites some of the disadvantages of the retroduodenal procedure, such as the danger of wounding a branch of the pancreatico-duodenal artery, and the danger of injury to the pancreas. He also refers to two cases reported by the Mayos, where attempts to remove stones from the lower end of the common duct resulted in forcing the finger well into the duodenum at an adjacent point rather than at the site of the papilla. The following case is reported for what it is worth in this contention:

Case IV.— Female, 32, one child, a patient of Dr. Young of Haverhill, had suffered for about eight years with recurring attacks of pain and tenderness in the region of the gall bladder, associated with fever and stomach disturbance. Three similar attacks within a year and a half, not including the last, had been followed by transient jaundice. Two weeks before the operation, an attack occurred in which deep jaundice quickly developed and persisted. Pain and tenderness not marked, pulse rapid, temperature irregular, no chills, no tumor, liver dullness much increased.

Operation:

Abdomen opened through outer border of right rectus. Liver much enlarged. Gall bladder, contracted and thickened, containing no stones. Foramen of Winslow obliterated by adhesions. There was no stone in the upper part of the common duct, but a hard mass, the size of an olive, could be felt behind the duodenum. The vertical portion of the gut was carefully isolated by gauze strips, and an incision about two inches long made in the anterior wall. The papilla was not seen, but the stone could be plainly

felt, just beneath the duodenum. I then cut directly down upon it, while the assistant made pressure from behind. It was easily scooped out of its bed, and was followed by a very profuse flow of bile. I was satisfied that I had opened that part of the common duct within the wall of the duodenum, because no distinction between the two structures could be made out; hence sutures were not necessary, and none were used. The anterior wall of the gut was closed by two rows of sutures and covered by an omental graft. The abdomen was closed in layers. Aside from persistent vomiting of bile for thirty-six hours, which was promptly relieved by lavage, the patient made an uninterrupted recovery.

Technique:

A sand bag is placed under the back opposite the liver, to bring the field of operation, especially the deeper parts of the common duct, as near the surface as possible. The incision is made through the outer border of the right rectus, from the costal margin downwards. I have found that upward rotation of the liver, after pulling it down from under the ribs, as suggested by Robson, to be of great assistance in gall bladder work.

My method of doing cholecystostomy is as follows: After the abdomen is opened, the neighborhood of the gall bladder is carefully protected by gauze. Two traction sutures are placed in the anterior wall near the fundus, about one-quarter inch apart, and an aspirator needle thrust in between them to evacuate as much of the contents as possible. The needle is withdrawn and an incision made at the point of puncture. The escaping bile or pus is quickly and carefully mopped up to avoid soiling. Gall stones, if present, are removed by a scoop, and if any are impacted in the cystic duct, they can usually be readily pressed back into the gall bladder.

A rubber tube is then fastened into the gall bladder by means of a plain catgut stitch through the edge of the incision and through the tube, and by a purse string suture of

chromic gut. When the latter is tightened the edge of the incision should be inverted; this makes a smooth joint and one not likely to leak. The gauzes used for walling off are removed and three moderate sized cigarette drains are placed in position,— one below the tube and one to either side of it. The abdomen is then closed generally in layers, up to the drains. I believe that tube drainage is much to be preferred to the old-fashioned way of sewing the gall bladder to the peritoneum,— a method often difficult, especially when the gall bladder is deeply placed, contracted, and more or less adherent, and one likely to be followed by a needlessly prolonged biliary fistula.

I am convinced, also, that the rubber tube possesses advantages over the double flanged glass tube tied into the gall bladder with catgut. In the case of the former, it comes away with the dressings after four or five days, when the fixation suture softens. On the other hand, the glass tube may become imbedded in the tissues and even require ether for its removal,— this happened in one of my own cases, and also in a case operated on by one of my friends.

Group IV.—Hernia.

There were 24 operations for hernia,—16 inguinal, 4 ventral, 2 femoral, and 2 umbilical.

Of the inguinal hernias 1 was double, 2 congenital, and 3 strangulated; there were 13 males and 2 females. One right sided hernia in a male proved to be the so-called sliding variety, the contents of the sac being cecum and appendix.

The only death in the hernia cases occurred in an old man with a right inguinal hernia, in whom strangulation had been present for several days. The intestine was gangrenous, and it was necessary to resect a portion six inches in length; anastomosis was made with the Murphy button. The patient died in five days from peritonitis.

In the first ten cases the Bassini method was closely fol-

lowed, except that kangaroo tendon was used to unite the conjoined tendon to Poupart's ligament, instead of silk, as is done by Bassini himself, and some other operators whom I have seen. In the last five cases the technique has been somewhat modified. Influenced by the teaching of Eccles, Scudder, Connell, Tinker, and others, I have not transplanted the cord. The idea that this step in the operation is unnecessary and may be omitted, is rapidly growing in favor. Furthermore, by leaving the cord in its natural position, certain disadvantages in the original procedure are eliminated. For example, as Connell has pointed out, the rough manipulation necessary in separating the mesocord from its normal attachments, and the added traumatism to which the cord is subjected in its new position, are inimical, in some degree at least, to the functional activity of the testicle, for, as is well known, orchitis, epididymitis, and even hydrocele and varicocele, do occasionally follow the Bassini operation. Scudder says: "It seems to me of comparatively little importance whether the cord is or is not transplanted. I am more and more inclined not to transplant it."

The only other modification which I have used is that of mobilizing the neck of the sac from behind the fovea inguinalis externa behind the internal oblique muscle, as described by Tinker. In this procedure the ends of the purse string are left long and threaded into needles which are carried through the internal oblique well above its lower border, from behind forwards; they are then drawn up and tied. This takes the neck of the sac away from the funnel-shaped opening into which the peritoneum normally falls, and seems to be of some advantage. In one recent case, where the neck of the sac was very wide, I found it much easier and safer in closing to use the continuous lock-stitch, as suggested by Scudder, instead of the ordinary purse string suture.

Another thing which helps one greatly in operating for hernia is a moderate Trendelenberg position. With the assistance of gravity thus afforded, the protrusion will often reduce itself, so to speak, after adhesions are separated, and, what is of far greater importance, it will remain out of the way while the operator closes the sac.

Femoral hernia is relatively uncommon, and there are but two cases in this list,—both strangulated. Of the various methods in general use I have selected the simplest, —the one adopted by Bull and considered by him as good as any,—that of stitching the pectineal fascia to the lower edge of Poupart's ligament with two or three kangaroo tendon sutures.

Both cases of umbilical hernia were in very obese females, over 70; the contents of the sac was largely omental, and strangulation was present in each. In these cases I used the Mayo technique, with overlapping from above downwards.

In ventral hernia it is my custom to take out a section of the abdominal wall which shall include the cicatrix. It is necessary to use extreme caution in these cases, on account of adhesions, which, although usually omental, may consist of intestine. Each layer is identified and separately approximated with kangaroo tendon of varying sizes.

Group V.—Miscellaneous.

In this group there were 31 operations for a great variety of conditions, viz: seven cases of intestinal obstruction, including three cases of post-operative occlusion by bands, two early and one late: no deaths; two cases of intussusception, one of which died after a second laparotomy for recurrence; one case of cancer of the sigmoid, in which resection and anastomosis with the Murphy button was done: death; one case of stricture of the descending colon, probably of tuberculous origin, in which perforation had

occurred, resulting in septic peritonitis: death; two cases of tubercular peritonitis: one death; two abdominal nephrectomies, one for disease and one for traumatic rupture of the kidney: no deaths; one case of gun shot wound of the abdomen: death; two colostomies for cancer of rectum with operative recovery in both; one case where a secondary operation was done for the purpose of draining a suppurating focus following removal of the adnexa: recovery; one case of infection and marked swelling of the mesenteric lymph glands with localized peritonitis of portion of intestine subtending area involved: recovery; one case of general septic peritonitis early in the puerperal period, supposed to be appendicitis or tube, where no lesion was found in either—probably a streptococcus infection: death; 14 explorations, including one case of acute hemorrhagic pancreatitis: death; cirrhosis of liver, two cases, two deaths; uterine fibroid associated with such extensive adnexal disease as to be considered inoperable, two cases; retro-peritoneal sarcoma, two cases, one death; pregnancy; inoperable cancer of abdominal wall and bladder; cancer of sigmoid; cancer of cecum; cancer of body of uterus with omental metastases and ascites; tumor of liver, not improbably angioma; typhoid fever supposed to be appendicitis.

A brief account of the fatal cases in this group may be of interest.

Three deaths followed operations for intestinal obstruction, the first from perforation and peritonitis associated with stricture of the descending colon, in a young man, probably of tuberculous origin. In this case mild and recurrent symptoms of obstruction had been present for several days before operation.

The second patient, a woman of 56, gave a history of three previous attacks of acute constipation with distention and abdominal pain, within a year. Each one was followed by bloody stools. Nine days before being admitted to the hospital she was taken with a similar attack, and continued to grow progressively worse, so that, at the time of opera-

tion, obstruction was practically complete. Upon opening the abdomen a tight annular stricture of the upper loop of the sigmoid was found, with marked induration of the gut for about an inch to either side of the constriction. The intestines above were greatly distended. I rapidly excised about six inches of intestine and did an end-to-end anastomosis with the Murphy button. The patient died on the fifth day, apparently from exhaustion, for the bowels had moved freely and the distention had disappeared.

The third fatal case in this list was a boy of four, with a history of diarrhœa for a few days, followed by abdominal pain, vomiting and bloody stools. On examination the abdomen was not particularly tender and not distended; a doughy mass was felt on the left side, extending from just below the level of the umbilicus upwards into the left hypochondrium. The invagination was found to begin at the middle of the transverse and to extend perhaps three inches beyond the splenic flexure into the descending colon. It was readily reduced by proximal compression. The patient did well for 48 hours, but at the end of that time pain and vomiting returned, and the bunch reappeared in the left side. The abdomen was reopened and reduction effected as readily as before; the gut looked well, there was no peritonitis. The patient died nine hours after the operation, presumably of shock.

In the list of explorations there were four deaths; one from shock, in a case of hypertrophic cirrhosis of the liver, associated with glycosuria, where marked tenderness and rigidity in the right upper quadrant, with fever and rapid pulse, were taken to mean an acute gall bladder; one from hemorrhage arising from œsophageal varices in a female suffering from cirrhosis of the liver, supposed to be gastric ulcer; one from acute hemorrhagic pancreatitis in a man 52 years old, of alcoholic habits and a previous history of gall-stone colic. He was taken 48 hours before the operation with severe epigastric pain, vomiting, distention and rigidity of the upper part of the abdomen and obstinate constipation; pulse small and very rapid, temperature 101.2, marked cyanosis. Acute pancreatitis was the probable

diagnosis. In this case I hoped to accomplish something by the method suggested by Hahn, and successfully practised by Woolsey, Pels-Leusden and others, of rapidly opening the abdomen, evacuating the septic fluid and draining. The patient was lightly etherized and an incision made in the epigastrium. The peritoneal cavity contained quite a quantity of brownish, dirty fluid, and spots of fat necrosis were found widely scattered over the omentum, thus confirming the diagnosis. The patient suddenly collapsed and died on the table, before infusion could be done. I then made an incision through the transverse meso-colon, exposing the pancreas. It was swollen throughout, and on section *in situ* showed the characteristic mottling of the acute hemorrhagic form of infection. I believe that it would have been much better in this case if I had infused before beginning, and had operated under local instead of general anæsthesia.

The other death in this group occurred in a female patient of 42 with a fixed tumor in the right iliac region, apparently the size of a large fist. It had caused her no pain, her only complaint being slight occasional soreness. Catamenia normal, nothing to be felt by vaginal examination. No loss of flesh, color fairly good.

Upon opening the abdomen, a large, irregularly globular mass was found on the right side, with the greater part of its bulk above the iliac border. The iliac vessels and ureter were seen coursing across the growth toward the inner side. Its surface presented many dilated and tortuous veins. I made a short incision through the peritoneum over the tumor, and from the ease with which it could be separated, I concluded that enucleation might be possible, although I realized that very free hemorrhage might occur. I therefore proceeded to shell out the mass as rapidly as possible. This was easily accomplished, except at its lower extremity posteriorly, where it seemed to be made up of

accessory nodular masses, so firmly adherent that separation was impossible, and I made no further effort to remove it. Gauze packing was not sufficient to entirely control the hemorrhage. Death occurred from shock two hours after the operation.

There was one fatal case of tubercular peritonitis in a little girl of ten years, where the abdomen was filled with bloody fluid.

One patient died of gunshot wound of the abdomen, who was operated on four hours after the injury. When first seen, just before the operation, there was no evidence of shock, pulse 86, of good quality, no pallor, and only slight pain in the abdomen. Examination showed a bullet wound on the left side, one inch below the level of the umbilicus and two inches from the median line. Under ether the wound was slightly enlarged and found to be penetrating. The abdomen was then opened by a median incision below the umbilicus. The peritoneal cavity contained a large quantity of blood; twelve perforations were found in the small intestine and four in the mesentery. The perforations in the mesentery and four of those in the ileum were closed by suture. A portion of ileum twelve inches long, containing eight perforations, was resected and end-to-end union made with the Murphy button. The abdomen was thoroughly flushed with large quantities of warm normal salt solution, a large wick was placed in either side of the pelvis, and the abdomen partly closed. During the operation two quarts of salt solution were given by intra-venous infusion. The patient died on the fifth day. Peritonitis was found at the autopsy. No further perforations were discovered, and all the closures, including the anastomosis, were intact.

The two cases in which abdominal nephrectomy was performed presented unusual features, and it may be worth while to report them somewhat in detail.

CASE I.—Charlotte C., 10, a patient of Dr. Young of Haverhill, ran into a step while sliding and struck upon her right side or back, at about 11 A. M., Feb. 19. Being, apparently, somewhat hurt and unable to walk, she was hauled on her sled by a playmate to the house of her aunt, who lived near by. She at once lay down on a sofa and remained there most of the afternoon, occasionally getting up and walking about, but making no complaint. In the evening she ate some supper and went to bed at the usual time, nothing being heard from her during the night. About 10 A.M. of the following day she was taken home by her father. They walked about a quarter of a mile, took an electric car, rode a mile, alighted and walked another quarter of a mile to her home. She was somewhat exhausted by the trip, but aside from this seemed to be in good condition. As the day wore on she complained of considerable pain and soreness in her right side, and Dr. Young was summoned. I saw her in consultation shortly after 5 P.M. At that time she showed a good face, somewhat flushed, pulse 150, temperature 100.2°, marked tenderness and board-like rigidity of the whole right side. There were no external evidences of injury. The bowels had moved three times since the accident, with no blood in the stools. The urine was passed normally and contained no blood. An exact diagnosis could not be made, but it was clearly evident that we were dealing with a serious condition, one which demanded immediate operation. The child was taken at once to the Hale Hospital. Operation 32 hours after the accident. An incision, three inches long, was made through the outer border of the right rectus, beginning just below the level of the umbilicus. Upon opening the peritoneal cavity, considerable free blood was found in the right iliac fossa and in the space external to the ascending colon. Blood had also escaped between the folds of the mesentery of the colon, dissecting its way half around the gut at some points, and near the hepatic flexure and ileo-cecal region it had completely encircled it under the peritoneum. A large retro-peritoneal hematoma was seen extending from the region of the right kidney well down over the pelvic brim. Upon breaking up and dislodging a mass of loose clot to the outer side of the colon, it was found that the peritoneum covering the anterior sur-

face of the kidney was torn, and fresh blood was seen pouring through the rent. This opening was quickly enlarged and the kidney palpated; it was partially detached and apparently extensively lacerated. I removed the kidney as rapidly as possible, tying the pedicle with a mass ligature. A gauze strip was tightly packed into the space occupied by the kidney and the abdomen partly closed. During the operation the patient was given a quart of normal salt solution by intra-venous infusion. Upon examining the kidney after removal it was found that the capsule and part of the cortex were torn off over a greater part of the postero-external surface, and that a laceration extended into the pelvis. The patient made an uninterrupted recovery.

Case II.—Female, 58, also a patient of Dr. Young of Haverhill, had suffered for several years from what had been supposed to be gall-stone trouble. She had had many attacks, in all of which vomiting and pain in the region of the gall bladder were present, and in some of which jaundice was also present. She had lost a good deal of flesh, complained of constant soreness in the upper part of the abdomen and suffered from persistent indigestion.

Operation:

Incision through the outer border of the right rectus from the costal margin downwards. A small cirrhotic kidney was found imbedded in a mass of adhesions, firmly attaching it to the right lobe of the liver, gall bladder and duodenum. It was removed with some difficulty, and after assuring myself that no stones were present in the gall bladder or common duct, I closed the abdomen. The patient made a good recovery, gained flesh rapidly, and has been in excellent health for three years.

In conclusion, the sum total of this experience shows a series of 281 abdominal sections with 31 deaths, a mortality of a little more than 11%. I have shown that some of the cases were operated upon under circumstances exceedingly unfavorable, and therefore it is fair to conclude that a considerable number of the deaths might have been prevented by more timely interference, especially in appendicitis. It is also fair to conclude that in the list of explor-

ations there were certain cases so unlikely to be helped by a surgical operation that none was indicated, and to avoid such occurrences one should carefully investigate every available source of information which might lead to a correct diagnosis. The point of view and the methods which I have used have been stated in the course of the paper, and need no further comment. It goes without saying that I have touched but lightly at a few points in the great field of abdominal surgery, and while such a limited experience gives no man the right to speak with authority, I would suggest to the suburban surgeon, that he must know his anatomy better, that he must be more careful of his asepsis, that he must use better judgment in the selection of his technical methods, that he must have the courage to refuse to operate simply because he has an opportunity, and finally to increase his resources in general he must make frequent visits to the surgical clinic of a large hospital and sit at the feet of the masters.

BIBLIOGRAPHY.

1. Bryant. Operative Surgery, Fourth Edition, page 998.
2. Connell. American Journal of the Medical Sciences. March, 1905.
3. Craig. International Clinics, Fourteenth Series, vol. I.
4. Elsberg. Medical Record, April 5th, 1902.
5. Fowler, J. Ryerson. Treatise on Appendicitis, 1900.
6. Greenough. Boston Medical and Surgical Journal, May 19, 1904.
7. Hahn. Deutsch Med. Woch, 190 V., page 5.
8. Kelly. American Medicine, April 20, 1901.
9. Lilienthal. Medical News, vol. 88, page 1020.
10. Meyer. American Medicine, April 12, 1902.
11. Morris. Medical Record, March 25, 1905.
12. Moynihan. Gall Stones and their Surgical Treatment, page 344.
13. Pels-Leusden. Deutsch Zeitschrift fur Chirug, September, 1903.
14. Richardson. Boston Medical and Surgical Journal, March 23, 1905
15. Routier. International Clinics, Thirteenth Series, vol. 3.
16. Scudder. Annals of Surgery, January, 1905.
17. Tinker. American Medicine, October 1, 1904.
18. Thienhaus. Annals of Surgery, December, 1902.

DISCUSSION.

Dr. S. J. MIXTER, of Boston: I feel I cannot let this paper go by without a word. I think Dr. Durant's results are good, and the paper is very good. But I want to begin and find fault with one thing. It seems to me that the serious fault is in regard to the title of his paper. The title is about the suburban surgeon. I do not understand why he should give it this title. There is not a thing that he has said that does not apply to the surgeon in the large city just as much as to the man whom he calls the suburbanite. A man is a surgeon or he is not a surgeon. There are mighty poor surgeons in the city, and there are mighty good surgeons in the country. Therefore I do not understand why he should give his paper that title.

As to his remarks on technique I have little to say. Let every man find out what is the best technique for him to use and let him use it. I think the men who are changing all the time fail to arrive at results. As far as the technique of asepsis goes, I am sure that we have all come down to simplifying methods rather than to elaborating them, and that to-day much less is expected if it is done carefully, if it is done thoroughly, than was expected years ago. After all, as Dr. Durant has shown and as we all know, the first important thing is the scrubbing brush and soap and hot water. What you use after that I think makes very little difference.

As I said a short time ago in speaking on the surgery of the stomach, a gastro-enterostomy and so on—let every man practise what he makes up his mind is the best method of operating in a given case and let him then follow it, but do not let him practise it until he has tried it on a living lower animal. I think this question of suburban surgery has solved itself. It is not like the old days when a few operations were done. Now there is more surgery than men connected in the hospitals can do. The only thing is that he should be a man who is fit for it and has studied it, and who, as Dr. Durant has said, does not operate simply because he has a chance.

Dr. E. W. CUSHING, of Boston: I feel somewhat as Dr. Mixter does as to the title of this paper. It does not

seem to cover the title. If it were entitled what the skilful operator is doing, it would answer the question more fully. In writing a paper some years ago I pointed out the growth of the small hospitals in America, especially in the New England States, and contrasted it with the hospitals of Germany, and found that in each one of these hospitals a great many mistakes had been made.

I wish Dr. Durant in closing would give us an idea, if he cares to, whether this is a fair representation of what the little hospitals and suburban surgery are at present.

In regard to his remarks on treatment, I think we should all agree. I have always taken the ground in regard to appendicitis that the salvation of the patient was in the very careful diagnosis and the very early operation. I understand that Ochsner's observations may have their bearing in cases which are not seen in the very beginning. It seems to me, however, that Ochsner's theory of waiting is not a very wise one, that on the whole it is better to try and make a diagnosis at once and operate at once even though it is found that the appendix is not very bad after all.

Dr. DURANT: In answer to Dr. Mixter's question in regard to suburban surgery, I would only say, that I wished to approach the subject in a modest way and report regarding two hundred laparotomies or thereabouts. In regard to the question as to the kind of work which is being done in the suburban hospitals, I have nothing to say. If Dr. Richardson were here the matter would be brought out more clearly.

ARTICLE IV.

CAUSES OF DISABILITY AFTER FRACTURES OF THE LOWER LEG AND ANKLE.

By FREDERIC J. COTTON, M.D.
OF BOSTON.

READ JUNE 13, 1905.

CAUSES OF DISABILITY AFTER FRACTURES OF THE LOWER LEG AND ANKLE.

THE thing we know least about in surgery, and particularly in minor surgery, is end results. This is particularly true in regard to fractures. The great experience gained in hospitals is after all a rather loose experience, and under present conditions of hospital practice (and abuse) in the large cities, no one man has a large series of fractures in private practice, and hospital cases are hard to follow. So we tend in prognosis to fall back on tradition and authority rather than to tabulate our actual experience.

I have never yet looked into the results of any single class of fractures without finding sharp discrepancies between the traditions and the facts as they stand. Many such discrepancies bear on prognosis and not particularly on treatment. I believe the facts that I shall here try to bring out call for some modification in treatment as well as prognosis. We were all taught, I think, that deformity was the thing we were to avoid in leg fractures — and taught little else. In fact, it seems to make little difference whether there is a little shortening, whether or no the much talked of "riding fragment" is present. Once solidly healed, the shaft is perfectly competent to bear weight whether smooth and regular or not — the important disabilities that follow these fractures are not of the leg but of the foot and ankle. Deformity as such is to be avoided, of course, but it is not of the greatest importance. I am firmly convinced that

the avoidable *disabilities* are nearly all due to one or more of three causes — as follows:

(*a*) Failure to maintain or restore the general long axis of the leg, whether local deformity is much or little.

(*b*) Mechanical damage to the ankle-joint or change in its plane, and

(*c*) Loss of joint motion from rigidity of muscles and tendons.

It is some years since we learned the lesson in regard to Colles' fracture that disability and deformity had no very close connection, and that over-long fixation was almost the only cause of functionally poor results. This does not apply directly in case of fractures of the leg and foot, because the leg has a function in weight-bearing as well as propulsion; hence the mechanical conditions of shaft and joint are more important, but when one comes to look at end-results the frequency with which disability is due mainly or entirely to stiffness is surprising.

I have been watching these cases for several years with a view to getting at the reasons for disability in individual cases, and about four years ago, with Dr. Bottomley, attempted a systematic study of end results of leg fractures by sending out cards to old patients. It did not work very well, owing largely to the fact that many leg fractures occur in young workmen of the drifting class. We got relatively few cases, and a recent search I have made found but few more. In the main the cases on which I have to base my conclusions are cases followed through the outpatient till they were walking, and more especially those who came to the City Hospital long after injury to seek relief from disability. In all I have notes of forty-seven cases followed to something like end results. They are in the main the *least favorable* of our results. Good results do not appear again in hospital. What the percentages are I do not know. My impression is that any consider-

able degree of disability is rather unusual. Most cases recover very well, though it takes a good while.

This is given as a preliminary paper, not because I expect to change conclusions, but because a study of this sort, to give considerable numbers of each type of fracture and each form of disability, should number hundreds of cases. Perhaps in time we may get such data. Only from careful detailed observation of large numbers, however, can we learn much; letters from patients as to results are almost worthless.

In the series studied the following conditions were found as causes of disability of greater or less grade:

a. Displacement of the bone ends, *per se*.

b. Change of axis of the leg.

c. Damage to joints, interfering with their bony mechanism.

d. Arthritis — "traumatic."

e. Static flat foot, not from deformity, but following over-use of atrophic muscles.

f. Contractures.

Shortening as a cause of disability I could never be certain of. Rarely is there over half an inch shortening, and unless it is considerably more than that I cannot see that any disability results, though there may be a slight limp.

Rotation out or in of the lower fragment I did not meet in more than a trifling degree. *Outward* rotation, unless slight, must be a favoring factor in producing "flat foot."

Poor circulation and persistent swelling often delay use, and so do damage. I have one case where the swelling itself, persisting for many months, seemed a *direct* cause of disability.

To take these matters up in detail:

As to the displacement of bone ends in fractures of the tibial and fibular shaft. Displacement is a factor in delay of union. I have watched very long course of union from

this, and have twice operated merely to quicken intolerably slow union by getting better apposition, but so far as direct result of *moderate* inaccuracy in replacing bone ends goes, I believe it is nil. If the deformity is *great* there seems to be enough bone strain to give some persistence of *local* pain and soreness on use. This was present in two cases of our series.

Displacement is, however, of importance in another way. The foot, to fulfil its function, should be under the weight bearing (vertical) line of the leg; a relatively slight bowing or overlap may throw it out enough to make trouble by increase of strain on ankle and foot. It is a question then not of bone outlines, but of preservation of the general axis of the leg.

The most common deformity seems to be in the direction of valgus. This throws the weight bearing line to the inner side of the foot. It may often act as does a rachitic knock-knee, and for the same reason, producing an ordinary "static" flat-foot deformity.

Deformity in the contrary direction if marked gives a defective tread and trouble in balance, and not infrequently some disability from the strain on the tendons outside the ankle. This deformity is a rare result in fracture of the leg, common as a result of fractures about the ankle. Deviation in this direction if of slight degree is a help rather than a damage, for it lessens the chance of a flat-foot strain —being in fact equivalent to the Trendelenberg operation for flat-foot.

Forward deviation is rare, except for the very mild deformity often associated with inward bowing where this is due to overlapping. Presumably the result of marked forward bowing would be like that resulting from like bowing after rickets — namely, a shambling gait without real disability. I have not seen it as a cause of disability after fracture.

Backward bowing, on the other hand, is common. This is because of the tendency of the limb to sag downward in its plaster or splints as the original swelling subsides, and because our inspection is apt to be limited to a front view of the leg which does not show this deformity.

Deviation in this direction if more than slight is rather serious. Study of cases show two factors that bear on this.

First, such deformity gives what is practically a shortening of the tendo-achillis, and moreover somewhat impedes the action of this tendon mechanically.

Second, the forward displacement of the foot results in (*a*) what amounts to an extreme hyperextension of the knee, (*b*) in a practical lengthening of the lever length of the foot, involving extra lift on the part of the soleus and gastrocnemius, already handicapped by the distortion of their tendon. The practical result is a considerable loss of working power and a good deal of clumsiness of gait. Not infrequently the disability is enough to warrant secondary operation. I have notes of two such cases operated and of one other that should have considered operation.

The distortions occurring from fractures involving the ankle joint bring in new elements. Here we have to deal not only with the change of axis but often with a widening of the joint or a dislocation of the astragalus.

Backward dislocation at the ankle complicating Pott's fracture is familiar to all of us. It results in partial loss of motion, in loss of any firm column of support, and sometimes gives almost total disability. Fortunately it is rare and probably always avoidable by proper treatment.

Forward dislocation at the ankle is less frequent. I have seen it but twice in the cases of this series, and in both cases associated not only with fracture of the malleoli but also with a chipping off of the front of the tibia at the ankle. Both were very lame, but one case had fibrous ankylosis,

the other much rigidity. There are no data to show how much trouble the displacement alone will cause.

Displacement inward has in this series been very common —this I take not to be the rule. Such displacement results from fracture across the tibia into the joint or from "inverted Pott's" fracture—in a mild degree and rarely from excessive correction of a Pott's fracture. The disability is from the malapposition of joint surfaces, from the twisted tread of the foot, and from associated stiffness. The disability is usually severe except where the distortion is from overcorrection in Pott's fracture, which does no harm.

Outward displacement is common, resulting in the rule from insufficient care in treating Pott's fractures, *i. e.* from neglecting the often needful extreme inversion of the foot. The symptoms are essentially those of static flat-foot plus muscle rigidity. Even slight deviation in this direction is apt to cause trouble. Where there is added a widening of the mortise as well as an outward distortion, we have an added factor of disturbance when static troubles begin.

The symptoms due to static trouble from ankle fracture vary, as do those of ordinary static flat-foot, according to individual equation; but I have notes of but one case with *marked* outward displacement due to fracture in the shaft where symptoms did not develop, and *no* such case where like deformity involving the ankle joint failed to give symptoms.

Sometimes with, sometimes without trouble with the arch we have a true arthritis. It is clinically characterized by swelling in some degree, by pain, sensitiveness and loss of motion, and inasmuch as these may all be due to other causes it is hard to be sure whether arthritis is present. In two of the cases of this series I know it was present because I operated on them—one showed fibrous ankylosis, the other marked loss of articular cartilage.

A comparatively large class are those in which with be-

ginning use relative overloading has overtaxed the muscles that should support the arch, but are unable to do their work, owing to the simple atrophy of disuse. These cases result in simple static flat-foot in all ways comparable to the ordinary orthopedic flat-foot with little or no tendency to the development of contractures.

By contractures I mean all loss of joint motion due to actual or practical shortening of muscle and tendon in the limb. Just what the essence of this change is no one knows; it seems to be the late stage corresponding to the early loss of motion common to all fractures, entirely recovered from in time in most instances, in others resulting in permanent change. It is not a pure disuse phenomenon, though averted by persistent early use. It has long been known that fixation, even long continued, has little and transient effect on normal limbs or on those affected with certain pathological processes, but this does not hold true in case of injury. In every case of fracture of the leg there is at the time that apparatus is removed some stiffness, associated with swelling and infiltration, but not proportionate to them. It is roughly, but very roughly, proportionate to the general amount of damage, *e. g.* it is much less with an uncomplicated fracture of the fibula at or near the joint than with a severe fracture of both bones. Its extent increases very definitely with the length of time the limb is held fixed. In cases where motion is begun early it subsides; in cases where motion is long postponed it often persists without change, or with little change, even after use. The site of the change is various, according to the site of injury—this is the most definite and most significant fact about it and I think helps to explain what it is we are dealing with. In cases of ankle fracture, or fracture below the middle of the leg, it is ankle stiffness with which we have to deal, while the knee is unaffected. If the break, on the other hand, is in the upper third of the shaft, the

stiffness at the ankle is far less, but there is a limitation of knee motion, often of considerable amount and tending to be permanent.

Throughout the cases I have looked into this has held true—it is a fact with which I was previously unacquainted and I think it helps our understanding of the character of the process. Where knee-stiffness follows fractures below the knee we can be sure that it has nothing to do with direct muscle damage or with alleged tendon adhesions. I am inclined to be skeptical about tendon adhesions. So far as the real evidence goes, they seem to be very rare. It must depend on reflex action or fixation, or both. Fixation alone should act equally on knee and ankle, for both are always fixed whether the fracture be low or high up, but this is not the case. On the other hand the cases studied show that those in which motion is long delayed give most stiffness. We must, I think, be forced to consider this stiffness due first to some reflex action (similar to spasm from joint damage) aggravated by long disuse and tending to pass over to a permanent shortening of the muscle. I believe this to be the only logical explanation.

Now as to the clinical side: if we are dealing with knee stiffness from high fracture we find limitation of motion definite but not extreme, and not greatly interfering with ordinary function. The limitation is of flexion only, for all fractures are put up in extension. At the ankle the problem is different. There is apt to be limitation in all directions — least in direction of plantar flexion, usually most obvious in limitation of pro and supination. If the foot has been put up without due care in securing a right angle, loss of dorsal flexion is considerable; if the right angle has been preserved, as it always should be, this loss is much less. In a general way short tendo Achillis is common to all fractures below the middle of the shaft; lateral limitation seems more in ankle fractures. Loss of

dorsal flexion entails according to its grade a difficulty in the normal stride. There is an attempt to compensate by medio-tarsal motion, and the patient suffers from what the orthopedists call medio-tarsal strain (familiar in other conditions involving short tendo Achillis).

Loss of lateral motion does not interfere with the straight step, but renders all accommodation to varying surfaces a strain on the shortened structures. These are the patients who "can walk all right in the house but not on the street," familiar to all of you. Every attempt to accommodate is a strain on the shortened tendon of the inner or outer side; every sharp strain brings its reaction of lameness, and often fresh access of stiffness — part of which remains permanently so that they get worse instead of better. Slight wrenches of these joints are serious in their results. This I have seen repeatedly when, after months of tolerable use, light fresh strains have resulted in considerable and permanent stiffness.

This stiffness differs from that of simple static irritation in that it is not relieved by rest, as are like types of foot of the usual flat-foot origin whether they show only pain or pain with spasm — nor are the cases we are considering greatly relieved by the wearing of carefully fitted supports — nor does the stretching of the Schaffer shoe limber them up to any serviceable extent. It is not well to be too dogmatic, but it seems that the pain and stiffness of these cases, with stiffness from trauma and disuse, are entirely different from the like symptoms in the cases above noted where mechanical disadvantage and weak muscles following fractures give a breaking down or laming of the arch later.

Dr. E. G. Brackett was kind enough to give some of these cases the benefit of his skill in treatment — and even in his skilful hands these really rigid ankles proved far more intractable than any rigid flatfoot; in fact they failed to improve to any degree under any treatment devised — while

the static cases following fracture yielded excellent results. Tenotomies we did not try, nor have I seen them tried in such cases save for the tendo Achillis; tenotomy of all the lateral tendons is a severe measure, though it would be amply justified by some of these cases if results could be promised.

Since I began to formulate this matter of contractures I have treated a number of leg fractures in house and out-patient departments at the hospital, by allowing very early a brief daily removal of apparatus and gentle active motion of the ankle joint. Passive motion I have avoided as more liable to do harm. In cases so treated I have had no trouble with position or with progress of union, and have had far more rapid return of mobility. Part of them only have been followed up, so I can not speak of statistics, either with the routine or with this method, but I believe this to be a step in the right direction. I am not ready to advocate the "massage" treatment of our French confreres, but I do think they have proved that we insist too absolutely on fixation in many cases, and that we fix fractures for a needlessly long time. Absolute fixation is necessary, not for a given number of weeks or until union is firm, but only in so far and so long as there is danger of displacement, — which is usually not long, — after this, protection with intervals of motion and perhaps massage to maintain flexibility and nutrition can do no harm. As said above we have learnt the lesson as it applies to Colles' fracture — the time has come when we must extend its use. Twenty years ago Sands' paper on the harmlessness of fixation was the beginning of the end of the old vicious practice of forced passive motion; we have gone to the other extreme now and have enforced absolute fixation beyond all reason.

To recapitulate, the chief causes of disability in leg and ankle fractures as they appeared in this series studied are:

Failure to preserve the line of the limb — and consequent static trouble.

Mechanical damage to the ankle joint.

Static trouble from over-load on atrophied muscles — apart from any mal-position.

Contractures.

In the first place the tendency to mal-position must be guarded against first by careful study of lesions and by careful reduction; secondly, by careful review (checked if may be by X-rays) of the result of reduction and of the present condition — a review to be made after not over two or three weeks when swelling and spasm are gone, yet while correction of mistakes is still easy. Later, operations are the only resort; undesirable, yet they are often of great service. The purely static troubles independent of malposition are usually readily relieved by careful plate-support.

The contractures are I believe avoidable in all cases unless there is long continued tendency to recurrence of deformity or delayed union or extreme trauma affecting soft parts as well as bone. I believe they call for treatment directed toward preserving flexibility and nutrition of the muscles, instituted before fixation has rendered the early spastic changes permanent. This means that rigid plaster bandages must be made removable at two to four weeks, not at three months, and that exercises without weight-bearing must be done persistently under proper supervision. If this is not done we must expect stiffness of ankles and less often of knees, as one of the great factors of disability. In five or ten years from today I believe any disability from stiffness due to contractures following ordinary leg fractures will be considered as great a reproach as obvious malposition is today, as great reflection on treatment as are the stiff hands and wrists after Colles' fractures — once so common — in the practice of today.

ARTICLE V.

EARLY DIAGNOSIS OF SURGICAL DISEASE IN THE URINARY ORGANS.

By BENJAMIN TENNEY, M.D.
OF BOSTON.

READ JUNE 13, 1905.

EARLY DIAGNOSIS OF SURGICAL DISEASE IN THE URINARY ORGANS.

For the purposes of this paper the urinary tract will be limited to the kidney, ureter, bladder and prostate. The prostate, although essentially a genital organ, when diseased interferes more with the urinary function than with the genital function, and must not be left out of account in any discussion of the urinary tract.

That early recognition of diseased conditions in these organs makes the desired "cure without undue risk, delay or pain" more often possible needs no argument, and to the physician who uses his trained brain, fingers and eyes, especially if he has access to a good microscope, there is little difficulty in recognizing the indications for further careful investigation of the urinary tract by the most modern methods of special diagnosis. Interesting as these special instruments are, and fascinating as is their use, this paper is to deal briefly and solely with the indications which should lead to the employment of one or more.

In order to obtain some idea of the frequency of diseases in these organs as compared with general surgical diseases, and to get some idea of the relative frequency of the different diseases, the annnal reports of the Massachusetts General and Boston City Hospitals have been taken for the last five years.

During this period there were over 35,000 cases in the surgical beds of the two institutions. More than 1100 of these are said to have presented some lesion of these organs, being about 3.1% of the total surgical admissions.

The prostatic diseases lead with 427 cases, the kidney follows with 373 cases, the bladder next with 304 cases, and the ureter last with 7 cases.

Most patients with disease in the urinary tract consult their physician on account of one or more of three conditions: First, Localized pain; second, Altered function; third, Hematuria. When all three are present there is hardly a doubt of the presence of some definite surgical condition in the urinary tract. That there may be some condition outside which reflexly excites symptoms, is indicated by a case recently seen with Dr. Mitchell of Portland, Maine.

He had done a complete and satisfactory perineal prostatectomy within a few days. The healing was perfect and urethra free, but the man continued to have pain referred to his bladder and urinated every half hour. Naturally blood and some pus were present in his urine. Finding tenderness over the right kidney and along the ureter, I suggested exploration, but both kidney and ureter were found to be normal.

At an autopsy two months later a bony tumor of the bodies of the lumbar vertebræ was found, not causing direct pressure on either kidney or ureter, but presumably irritating the nerve supply of the bladder. This combination of conditions is so very rare that it may be disregarded in making a preliminary diagnosis.

When any two of these conditions are found, I believe that all possible surgical conditions in the urinary tract should be ruled out before attention is centered on other organs, or the patient is left to Nature somewhat upset by the various so-called urinary antiseptics and sedatives.

Of the single symptoms *Pain* is of little value in directing attention to the urinary tract, and the *great majority* of pains without disturbance of function or blood in the urine will be found to be caused by some other organ or system.

Disturbance of function is almost worthy of being called pathognomonic. The normal adult individual under average conditions of heat, excitement and drinking, empties his bladder from four to seven times a day, and should be able to sleep seven or eight hours undisturbed. There should be no difficulty, discomfort, or urgency about the act. Regular variations from these habits are the first of all symptoms to appear in several urinary diseases and must be inquired into carefully and regarded with grave suspicion when persistently present.

Hematuria always means something wrong in the urinary tract, and when persistent or recurring intermittently should lead to careful study as to its source and cause, not because the loss of blood is always dangerous, although it may hasten the end in some conditions, but because it is almost invariably an indication of some serious and possibly curable condition.

It is possible for a patient to have some surgical disorder of the urinary tract in which none of these symptoms appear for months, but under continuous observation something will be found if these organs be kept in mind.

So far as the writer has been able to look up records, there is only one of these surgical conditions which may not be found at any age. Infants and children have been reported as having movable kidneys, calculi, malignant growths, and in fact all of these diseases except prostatic hypertrophy.

Beyond the fact that most of the movable kidneys recorded are female, and all the hypertrophied prostates male, either sex may suffer from any of these conditions.

Instead of discussing the symptoms of each disease separately, I have grouped the most important symptoms and attempted to show why they appear under different conditions.

Pain from disease of the *kidney* may be felt at the point

of injury or referred to the opposite side or to some distant point. Any mechanical condition which interferes with the outflow of urine, such as a kink in the ureter, or stone, or clot, or swollen lining, or tumor pressure, or stricture, or ligature, which prevents the free flow of urine from the kidney, will cause pain, and the same sort of pain with additions according to the location and character of the obstruction. The same sort of pain may be felt in certain infections of the kidney in which the bulk of tissue within the capsule is increased, or from hemorrhage under the capsule, or after artificial distention of the pelvis.

As has been previously pointed out, there are some observations which go to show that the kidney is extremely tolerant of all conditions which do not stretch its capsule, and that simple slitting of this capsule will for a time relieve the pain of pelvic distention, even when caused by stone in the ureter.

How much if any sensation is possessed by the kidney beyond its reaction to capsular stretching, or what is sometimes called internal pressure, I do not know.

Many malignant growths cause no pain for a long time, especially in children. The same is true of some renal calculi, but just as soon as a blood clot or fragment of tissue, or bit of calculus, plugs the ureter, the typical attack of pain begins.

Palpation of a normal kidney, even if considerable force be used, causes no pain; packing, washing out or removing drains in the pelvis, causes no pain in an exposed but uninfected kidney. Possibly its only sensory nerves lie in the capsule or walls of the pelvis. Of this we ought to know more.

If there is any characteristic of pains arising in the kidney, it is their radiating character. They may be referred to any point between the shoulder and the ankle on the same side, to the region of the opposite kidney, to the

pelvis, or to the abdomen generally, and all at the same time.

Pain in the *ureter* is produced by stretching or distention. Passing a ureteral catheter into a normal ureter, even if purulent urine is flowing through it, causes less pain than passing a soft rubber catheter into a urethra. Apparently there is little sensation except that of tension in any case, but this may become extreme pain when a concretion is being forced down by the peristaltic contractions.

There is no possibility of being sure whether a pain which a patient describes is due to a renal or ureteral condition without other evidence. His sensations may be precisely the same in either case.

The *bladder* is not sensitive unless inflamed and then it tolerates catheters and drainage tubes which are not moved about. Most of us are aware of the location of the dull ache of an acutely distended bladder, yet many patients carry from a few ounces to some pints of urine constantly without being conscious of it. From one woman who was not suffering very greatly, I drew six quarts and half a pint of urine within one-half hour, and this is much below the greatest record.

Some cases of tuberculosis and new growths in the bladder suffer very little during the early stages when radical treatment may be expected to produce the best results. One of my patients with cancer of the bladder had pain only on urinating until the growth reached a size to mechanically obstruct the urethral orifice. Many calculus cases are reported to have suffered little until the bladder mucosa became damaged and infected.

Prostatic cases suffer pain like kidney cases from stretching of the capsule in inflammatory conditions and from the secondary changes which occur in the obstructed bladders. Pain from prostatic trouble alone is usually referred to the rectum, glans penis, or perineum. When the bladder is

involved there is the local discomfort before referred to, and often the almost constant tenesmus due to irritation at the orifice of the bladder and in the prostatic urethra which may amount to pain.

Of the diseases presented in the cases from our two hospitals those representing acute infections are most likely to cause constant pain; those representing or producing foreign bodies such as calculi or clots of blood or pus or good sized fragments of tissue, almost always produce intermittent pain, while the new growths may be painless for years. In a general way, pain is more likely to be referred to a point below its origin than above it, but disease of the lower part of the tract is so likely to lead to infection or painful distention above it, that there is always a possibility of being led astray.

Altered Function.

Somewhere I have read that the act of urination begins with a pull of the ureteral muscle fibres which bound the trigone and are continued into the veru of the prostatic urethra, or a corresponding point in the female urethra. This pull opens the internal orifice and allows some fluid to trickle into the urethra which first makes us conscious of its presence. The voluntary sphincter may now contract and force the fluid back for a time or relax and allow it to pass on. If this description be correct, and I know of no anatomical, experimental or personal reason for doubting it, it explains how function may be altered by conditions remote from the bladder.

Frequency may be increased by any condition which diminishes the capacity of the bladder or causes irritation of these lower ureteral fibres. Therefore in old cystitis, when the capacity may be reduced to a few cubic centimeters, in tumor and calculus cases with constant irritation of the trigone, and especially in the occasional acute

inflammations which accompany an enlarging median lobe of the prostate and which directly affect these fibres, we may expect increased frequency. To explain its occurrence in renal and ureteral conditions we have only to remember the continuity of the ureteral fibres from kidney pelvis to verumontanum or the corresponding point in the female urethra. We have all known the frequent dribbling overflow of a distended bladder to be mistaken and treated—or neglected—as an "inflammation of the bladder" or a little "kidney trouble." There is no excuse for a physician's making an error of this sort. We have no right to guess when we can find out.

Urgency. Some individuals find it impossible to resist the impulse to urinate. If they attempt to prevent the flow, they leak. In every case there is an uncontrollable contraction of the bladder, due possibly to irritation of the lower ureteral fibres, to a calculus in the ureter or bladder, a new growth in the bladder, an enlarging median lobe of the prostate or an inflamed condition of the urethra nearest the bladder outlet, or else an incompetent voluntary sphincter, any of which conditions may disturb the normal relation between bladder capacity and contraction. I have seen examples of all the above conditions, and doubtless there are others that will produce the same symptom. It is too suggestive to be neglected and must bear an intimate relation to

Incontinence, which, except in the overflow of distended bladders and as it may suggest search for and removal of all irritations wherever found, has no surgical significance in this paper.

Difficulty in urination, perhaps as common a symptom as any, results from either mechanical obstruction or some fault in the nerve supply. It occurs in conscious patients with paralysis of the abdominal muscles and competent sphincter, as well as in those who are in a comatose or delirious condition. My limited observation has led me to

the belief that paralytic and comatose patients are quite as apt to be incontinent as to have retention, unless they have some obstacle which they had previously been able to overcome by voluntary abdominal pressure.

The mechanical obstruction of which we hear most today is the hypertrophied prostate; and while unable to discuss it as I should like, I would remind you that it is not a disease of advanced life exclusively. Several cases have been reported in the thirties, and between forty and forty-five the number of men whose prostate begins to produce some discomfort and residual urine is very considerable.

Anuria may occur in the course of certain acute diseases, as a result of some forms of poisoning, after trauma to the urinary tract below the kidney or to the kidney itself, from vaguely understood reflex disturbances outside the urinary tract, and from any condition which mechanically obstructs the flow of urine into the bladder. The last group of cases is not always easy of separation from the others. Fortunately patients usually go from two to five days before symptoms of uremia appear, which allows sufficient time, if promptly used, for decision and operative measures if thought proper. Absence of urine in the bladder during twelve consecutive hours, if proved by a catheter, may well arouse grave apprehension, and twenty-four hours is a limit beyond which the responsibility lies very heavily on any single practitioner.

Hematuria.

Red blood may show in the urine in amounts recognizable to the naked eye, with or without clots, or in such quantity as only to be recognized by the aid of the microscope. In either case it may be intermittent in appearance, but in the first case blood is likely to be found on microscopic search long after it has disappeared in gross.

While it may come from any part of the urinary tract and from several causes, persistent or recurring hematuria is so strongly suggestive of a new growth that, next to anuria, it is the gravest single symptom of all.

According to Albaran, hemorrhage was the first symptom in 78% of 140 malignant bladder tumors and was present at some period in nearly all.

According to Israel 70% of 66 malignant renal tumors were first indicated by hemorrhage, and 93% showed it at some period.

Deneclara in 168 cases of tumor of the kidney in the adult found that hemorrhage was the first symptom in 69%.

Even under exceptionally favorable conditions the cause and source of the hemorrhage may remain in doubt for years.

A recent patient of mine had hematuria for three years without renal symptoms or palpable tumor. There was no loss of weight or strength, and he was particularly active physically. He had a slowly growing prostate with considerable swelling and redness of the median portion as seen through a cystoscope which was supposed by one of our most experienced men as well as by myself to be the source of the hemorrhage. A third surgeon seeing him six months later found a distinct renal tumor and a hypernephroma at operation. This experience has taught me never to accept a probable cause of hematuria without ruling out other possible causes.

While it may possibly be due to some harmless condition, its almost constant association with malignant disease and calculus make it to my mind quite as grave, though not so urgent, a symptom as anuria of a few hours' duration.

All writers agree that blood in the urine appearing without reference to any physical violence, exertion or jarring, is very suggestive of new growths. When the hematuria

follows such violence, even if slight, they consider it more probably from the presence of a calculus. In either case it is a hematuria.

If there be also a pyuria the diagnosis is complicated by an infection which may wholly account for the hematuria, or which may be a secondary process grafted on to the original disease.

This brings us to the most frequent of all urinary conditions—continuous or recurring discomfort rather than pain, often poorly localized, little if any disturbance of function, little if any blood in the urine, but pus in the bladder urine continuously in varying amounts. The majority of cases presenting these symptoms can be cared for by any man who will handle a catheter and his solutions in a cleanly way; many will recover if they will wash themselves out by drinking enough water, but the other sort who continue in about the same condition for months, swallowing everything from hexa-methyline tetramine to Seng, are the patients who appear later with incurable tuberculosis, or inoperable cancer of bladder, ureter or kidney, with trabeculated and rotten bladders, with kidneys ruined by calculus obstruction, or honey-combed with pus, or transformed into that puzzle of the pathologist—amyloid. These are the cases which arouse regret in the mind of the surgeon — or the pathologist.

If these symptoms will disappear at all under irrigation and mono- or poly-syllabic treatment, they will go within a few weeks at most. If they persist beyond the few weeks, either the infection is not reached by the bladder irrigation or controlled by the chemicals, or there is an infection in the deeper tissues, or else the pus is masking some condition like a calculus or a new growth which is entirely distinct from the infection, though it may be the predisposing cause.

This is the time to study the case by every available

method until the condition is understood and proper measures and time for treatment are determined. A case neglected at this stage may get well from a colon bacillus pyelitis or pass a small calculus through the natural channels, or possibly make a spontaneous recovery from a tubercular infection; but who can tell which case?

Of pyelitis Kelley of Baltimore says in substance: "It is of the utmost importance, but is rarely recognized in the earlier stages, or when found is often thought unworthy of attention because not immediately provocative of serious symptoms.

"The kidney is an organ more necessary than the uterus or Fallopian tubes, but there is a general indifference to mild cases of pyelitis while the large pus kidneys are noticed.

"It is important to recognize any grade, because the severe cases may result from the mild."

Pyelitis, pyelo nephrosis, pyonephrosis, perinephritic abscess, all infective processes, cover one-fifth of all surgical kidney admissions to the two hospitals. If to these we add the cases of tubercular infection which may also show with early purulent urine we take in nearly one-third of the surgical kidneys.

If we follow modern theories and include the calculus cases as caused by an earlier inflammatory condition which might possibly have been recognized, we find that one-half of these surgical kidneys might have been suspected in an early stage from the presence of pus in the urine, and possibly spared some suffering at least.

Inflammatory processes in the bladder are likely to be recognized earlier because of the alteration in function which nearly always accompanies them, and because of the pain which also impresses the patient with the idea that something is wrong.

The inflammatory condition which persists longest without symptom is found in prostatic hypertrophy. Often

nothing is noticed by the patient until he suddenly realizes that he has to rise one or more times at night to empty his bladder. This symptom in a man over forty is always suggestive and characteristic of a bladder with residual urine.

Of the general symptoms I will only remind you that patients with surgical disease of one kidney are apt to sleep on the affected side; that varicocele may develope on the same side as a kidney tumor; that kidneys with acutely distended pelves and capsules are exquisitely tender, while hydronephrotic kidneys and some very large tumors may be little sensitive; that rectal or vaginal touch may discover tenderness over the lower end of one ureter, indicating trouble higher up on that side; that median obstruction in the prostate is not always to be felt by the rectum; that a tender "Israel's point" — junction of twelfth rib with outer border of erector spinæ — is valuable in identifying the kidney as a source of vague abdominal pain, and that the normal kidney, ureter, bladder and prostate are not painful under reasonable pressure.

Localized pain, altered function, hematuria, with or without pus — these are the cardinal symptoms of disease in the urinary tract. Tumor, tenderness, and residual urine do not require complicated apparatus for their recognition, and their presence or absence can be determined by any physician who will take the pains to examine. These preliminary points are simple, sufficient for a correct diagnosis sometimes, and always trustworthy in indicating the urinary tract as a source of trouble. Nothing in surgery requires more concentrated attention, more accurate observation, or a finer technique, than a complete and correct pre-operative diagnosis of the more obscure conditions in the urinary tract which stand between our patients and health.

SURGICAL DISEASES OF THE URINARY TRACT, B. C. H. AND M. G. H. 1900—1905.

KIDNEY.

Inflammatory	Pyelitis	13	191 = 51%
	Pyonephrosis	33	
	Perinephritic abscess	26	
	Tuberculosis	46	
	Calculus	73	
Mechanical	Hydronephrosis	27	142 = 38%
	Moveable	89	
	Rupture	26	
New growth	Sarcoma	19	35 = 9%
	Carcinoma	6	
	Hypernephroma	7	
	Adenoma	1	
	Cystadenoma	1	
	Neoplasm	1	
Other disease			5
			373

URETER.

Inflammatory	Tuberculosis	1	3
	Abscess	1	
	Ureteritis	1	
Mechanical	Stricture	2	4
	Calculus	2	
New growth			0
			7

BLADDER.

Inflammatory	Cystitis	100	124 = 41%
	Tuberculosis	24	
Mechanical	Calculus		98 = 32%
New growth	Carcinoma	29	41 = 13%
	Papilloma	11	
	Sarcoma	1	
Other disease			41
			304

PROSTATE.

Inflammatory	Hypertrophy	329	392 = 91%
	Prostatitis	40	
	Abscess	20	
	Tuberculosis	3	
Mechanical			0
New growth	Carcinoma	26	33 = 8%
	Adenoma	4	
	Sarcoma	3	
Other disease			2
			427

DISCUSSION.

Dr. S. J. MIXTER, of Boston: Dr. Tenney has asked me to discuss his paper. The ground has been so well covered that there is very little left to be discussed. The key note of his paper is that all the symptoms of a disease should be studied with every possible means in our power. Now that is a very important thing. One sees so many cases of renal, cystic or other disease of the urinary tract go on year after year, having been treated as a case of this, that or the other, given citrate of potash and all the various things without the slightest result, without the slightest scientific search having been made for the real cause. At the same time his paper points to another suggestion, which is that given a case appearing for the first time with some urinary symptoms, all these things should not be done at once. Years back, when anything was the matter with anyone, he would come to the hospital, and every patient who came into the hospital with any urinary trouble, had everything possible done to him at once. In a great many of these cases it seems to me absolutely unnecessary. In the average case I think that all these things should be gone at slowly and in turn, a sound or catheter not passed without it is absolutely necessary after a careful examination of the patient's urine and symptoms.

There are one or two points about which I wish to speak. One is the renal question to which he refers. I think every surgeon who has much to do with renal surgery has seen cases with severe pain in the kidneys, and a stone is suspected. Even in some cases we have hematuria, and there has been no question in the surgeon's mind but what there is a stone in the kidney, but after opening the pelvis he finds no stone and the symptoms disappear. This is something which every one has noticed, and it is a thing which has been applied. It is the reason why the operation of splitting the capsule for calculus in the kidney has been advocated by men like Johnson and others.

There is one point in the diagnosis of renal calculus apart from the X-ray that has been useful to me a number of times, and is mentioned either by an Englishman or an Irishman, I have forgotten which, who spoke of it as al-

most constantly present, that is the sense of pain and relative tenderness over the kidney or ureter where strong percussion, like a blow of the fist, has been applied. When it is present I think it is a very significant symptom.

Dr. O. C. SMITH of Hartford, Conn., spoke a few words in favor of skiagraphy, and said he thought it should be employed in every case where there is a suggestion of stone or any growth of the kidney. He further said that he has found it the greatest aid in his early diagnosis in these cases. He said he has advocated it in many of the small cities, and remarked that several men are making a study of it.

ARTICLE VI.

EMPYEMA OF THE FRONTAL SINUS.

By FREDERIC C. COBB, M.D.
OF BOSTON.

READ JUNE 13, 1905.

EMPYEMA OF THE FRONTAL SINUS.

THE subject of this paper is empyema of the frontal sinus. I shall not trouble you with any history of the disease, but will begin at once with the symptoms. One of the most important of these is headache, which varies in character in various cases, and even in the same case. Ordinarily the headache begins in the morning and lasts for three or four hours, ceasing about noon. It may, however, in some cases come on in the afternoon and last in the same spasmodic way for two or three hours, and then cease. Theoretically, the cessation of the headache should be accompanied by a free discharge of pus from the nose; but practically this is not always observed. The headache is evidently the result of pressure caused by pus in the frontal sinus, and its appearance in the morning is perhaps due to the fact that during the recumbent position the ostium of the sinus is not subjected to the same pressure that it is in the erect position, hence the collection of pus becomes greater in the morning.

The degree of pain varies in intensity, according to the pressure of fluid within the sinus, from a feeling of weight or pressure over the forehead to a dull pain, or even an intense pain. The character of the pain is described as beating or pulsating, but may be simply a dull ache or an acute neuralgia. Nor is it unusual to have frontal sinusitis without any pain, the whole question depending upon the pressure in the sinus.

And an even more variable symptom is tenderness over the sinus, which may be felt on pressing the upper wall of the orbit, taking care to avoid the supraorbital nerve.

The second symptom of importance is discharge from the nostrils. From the frontal sinus the discharge is not ordinarily very profuse. It may be yellow or greenish yellow, and alternate between very thick and quite thin. An odor disagreeable to the patient's perception, but not always so to the family or physician, is at times present, and a tendency to crusting or scabbing is very noticeable.

The amount of the discharge is subject to variations, depending upon the clearness of the nasal frontal duct, and when the pain, such as I have said is caused by the stoppage of the duct, is most severe the discharge should be less, and vice versa. It is here to be observed that the discharge resulting from the frontal sinus may flow either anteriorly or into the nasal pharynx, and this depends upon the position of the head or on various conditions within the nostril. It will be seen at once that this fact makes an estimate of the amount of discharge very uncertain.

A third symptom is stoppage of the nose. This is also a somewhat uncertain symptom, and is usually caused by irritation of the pus discharge. This irritation results in an hypertrophy of the turbinated tissue, over which the pus passes, or in polypoid formation, producing an increase in the thickness of the normal tissues. From the pus discharge results also an eczema of the edge of the nostril in many cases, which if unilatural is always significant of sinus disease.

We have mentioned in dealing with the symptoms of frontal disease some that are characteristic of lesions of this particular sinus, but others which, while occurring in this disease, might nevertheless at times be due to other neighboring sinuses. When the anatomical preparations are inspected it will be seen how great are the difficulties in diagnosis. The antrum, anterior ethmoidal cells and the frontal sinus open into a common infundibulum or canal, the opening of which is covered by the middle turbinate

bone. Pus appearing under this middle turbinate can come from any one of these cavities. Even after ablation of the anterior end of the middle turbinate it is usually impossible by watching the discharge to ascertain from which sinus it comes, because some of the anterior ethmoid cells are so high up as to be out of sight intra-nasally. For our diagnosis of the origin of the suppuration we must therefore depend upon our ability to ascertain which of the cavities contains pus. Even when this is ascertained our difficulties are not yet over, for in the last few years it has been proved that the antrum may contain pus in quantity and nevertheless may not originate it, but simply serve as a reservoir for other cavities. This may seem too natural to require comment, and yet many antra have been operated again and again by every radical operation known to surgery, and with constant disappointment, owing to failure to recognize this fact. If this is true of the antrum it may also be true of some of the other sinuses, that pus existing in a cavity does not necessarily mean pus originating from that cavity. The means at hand of ascertaining whether or not the frontal sinus contains pus are as follows:

Externally, pain or pressure over the floor of the frontal sinus or its walls. This of course only applies to acute conditions in the sinus. It is important in this test not to press upon the supra-orbital nerve, but rather on the outer portion of the orbit.

Examination of the nose may show pus under the middle turbinate. If so, it probably comes from the frontal, ethmoid or antrum. If no pus is seen the following methods may be used. Logan Turner advocates causing a vacuum with a Politzer bag, allowing the patient to swallow at the same time, thus producing a vacuum and drawing the pus down into the nostril. Or, if possible, a catheter may be passed into the sinus and air can be blown in by a Politzer bag, displacing the pus in the sinus which at once appears

in the region of the middle turbinate. Transillumination of the antrum gives us further information. If both antra are bright we can feel sure the suppuration is in ethmoid or frontal. If the antrum on the affected side be dark it probably contains pus, and if a trocar be introduced through the lower meatus and the pus washed out it will reappear in a short space of time if it comes from the sinuses above the antrum; whereas if it originates in the antrum, it will take a considerable time to fill the antrum again.

Transillumination of the frontal will in some cases show disease there, but often fails to do so, as the light passes through the skin. An opening in the skin may be made under the eyebrow and a small opening into the sinus trephined; in case pus is found to be present, the opening can be enlarged and the sinus cleared of pus and granulations. A small opening in the locality leaves only a slight scar.

Treatment.

Hajek recommends intra-nasal removal of obstruction in the fronto-nasal duct, and states that he believes this to be a procedure of great value. It goes without saying that polypi and swelling of the middle turbinate should be removed, and the middle turbinate or its anterior end, to afford free drainage. My experience however is that this procedure seldom cures chronic cases, although it may afford relief. So far as I know, most surgeons have abandoned it in favor of the more radical external opening.

Radical operations are as follows:

1. Method of Nebinger-Praun. The incision goes from the naso frontal suture at the edge of the orbit to a point over the supra-orbital notch. The anterior bony wall is chiseled away, then a vertical cut four to six centimetres long is made, following the frontal vein. After opening, the contents of the sinus is wiped out and the mucous

membrane scraped. The naso frontal duct is then widened and curetted. The drainage of the frontal is secured by a wick at the base of the wound.

2. Method of Ogsten-Luc. An incision at the inner third of the edge of the orbit, which may be lengthened either outwards or towards the root of the nose. The periosteum is pushed upwards and outwards towards the orbit. The opening of the sinus is a little to the outer side of the median line. The nasal-frontal duct is curetted as well as the interior of the sinus and some of the anterior cells of the ethmoid. Through the wide communication with the nose thus made, a drainage tube is put in and the outer wound closed. After eight days the bandages are removed. First drainage tubes were used between sinus and nose, but these were later replaced by strips of iodoform gauze.

3. Method of Kuhnt. In this method the whole of the anterior wall of the sinus is removed with its diseased mucous membrane, as also the upper part of nasal frontal canal. The incision is as follows: a horizontal incision along the supra-orbital margin from the eye brow to the outer third of the edge of the orbit; then a vertical incision from the inner edge of the wound through the soft parts of the periosteum. After lifting the soft parts and the periosteum the whole wall of the sinus is removed with chisel and bone forceps and the edges of the wound are smoothed. All projections into the cavity are thoroughly removed, the mucous membrane is most carefully scraped from the sinus and from the upper part of the duct. The wound is sewed up, leaving only room for a large drainage tube. This method has met with fair success, but causes considerable depression and does not remove the anterior ethmoid cells. When the disease is bilateral, the cosmetic result is of course better.

4. Jansen's method. Removal of the lower wall of the sinus and resection of the lower border of the anterior

wall; curetting the mucous membrane of the sinus and of the granulation tissue. This method is not at present used by Jansen, but in 1903 he was doing a bone-flap operation of very much the same character as that practised by Hajek.

5. Method of Ridell. Removal of the anterior and lower wall of the sinus. Thorough and satisfactory except for the great deformity.

6. Killian's method. Incision as in Ogsten-Luc, with supplementary incision beginning at root of nose and passing under inner edge of orbit. Incision is not carried through the periosteum of the arch of the eyebrow, but the soft parts are lifted and incision through the periosteum made a little higher up so as to preserve that of the arch. Removal of the whole anterior wall of the sinus with curetting mucous membrane and all inequalities of the bone. Incision then made in the line below orbit as described. Ethmoid cells entered through nasal process of maxillary bone and thoroughly curetted. Upper wall of orbit removed, allowing orbital fat to fill up depression caused by removal of obital wall. Closure of wound without drainge.

7. Coakley's method. Incision, as in Ogsten-Luc operation. Cavity thoroughly curretted with the assistance of electric lights placed in sinus. Nasal duct curretted but not widened, the object being to secure its closure; sinus then packed with gauze and repacked until it heals from the bottom.

The advantages and disadvantages of these methods may be summed up in a few words. The Ogsten-Luc has for advantage the slight deformity produced. For disadvantage it has the great probability of recurrence. The same is true of Jansen's method. Kuhnt's, Ridell's and Coakley's methods yield less probability of recurrence, as they destroy the sinus, but they produce greater or less facial deformity; Coakley's probably giving the least of the three. To all of these methods there is one great objection—that

the ethmoid cells are not sufficiently considered. Killian's operation seems to combine the advantage of sinus obliteration with the removal of the ethmoid cells; but it is hard to judge as yet of its practical results as far as deformity and danger to the eye are concerned.

ARTICLE VII.

THE APPLIED ANATOMY OF THE FRONTAL SINUS.

By HARRIS P. MOSHER, M.D.
OF BOSTON.

READ JUNE 13, 1905.

THE APPLIED ANATOMY OF THE FRONTAL SINUS.

THERE are two frontal sinuses, a right and a left. They are separated by a median partition which corresponds to the sagittal suture of the skull. The sinuses are placed in the middle and inferior part of the anterior face of the frontal bone. Each sinus represents the termination of the anterior part of the ethmoidal labyrinth of which it is an off-shoot. The sinuses stretch like wings transversely over the anterior portion of the orbit. They can be represented diagramatically by drawing an arc which has for its centre the mid-point of the base of the nasal bones and extends from the middle of one supra orbital ridge to the middle of the other.

A horizontal section of the skull shows that the sinuses have the form of a triangular pyramid. The base of the pyramid is in the middle line while the apex extends outwards a variable distance over the orbit. There are, therefore, three sides or faces to the sinus, the anterior face which is sub-cutaneous, the posterior or cerebral face and the inferior or orbito-nasal face. In the posterior inferior angle of the pyramid, the width of the corresponding half the ethmoidal notch from the median septum there is an opening which connects the cavity of the sinus with the nose.

All recent studies tend to demonstrate that the frontal sinuses are ethmoidal cells which have insinuated themselves between the two tables of the frontal bone. The following facts support this view: The development of

the sinuses corresponds in time with that of the ethmoid cells. The sinuses are not present at birth but begin to appear toward the end of the first year or during the second. They spring from the level of the anterior part of the ethmoidal labyrinth, and enlarge progressively upwards and from within outwards. When the sinuses are small they can be distinguished from the neighboring ethmoidal cells only by the fact that they are found between the two layers of the frontal bone. Further, the sinuses open indifferently into the two grooves at the top of the middle meatus, into which the ethmoid cells open, and the ethmoidal cells often mound into the floor of the sinus. Finally, upon the whole length of the ethmoidal notch of the frontal bone there is a row of little half cells which is matched and completed by a similar row of cells upon the upper surface of the lateral mass of the ethmoid bone.

The Size of the Sinus.—According to certain writers it is possible to estimate the size of the sinus by the prominence of the superciliary ridge. Roughly this is true. The sinus does not begin to develop until after birth and does not reach its full size until between twenty and twenty-five years. Defective development of the sinus is characteristic of certain negro tribes. At eight years of age the sinus measures 6-7mm (¼in.) The average capacity of the sinus ranges between 4-5cc (1-1½ drachms). Owing to more or less deviation of the septum which divides the sinuses they are seldom of the same size.

The Mucous Membrane of the Sinus.—The mucous membrane is a derivative of the mucous membrane of the nose and has the same extensive vascular and nerve supply. The mucous membrane of the nasal fossa consists of two kinds, the respiratory and the olfactory. The mucous membrane of the respiratory region is distinguished from that of the olfactory region by its thickness (4mm) and by the presence of a venous network of large size. The epi-

thelium is ciliated and of the stratified columnar type. Within its superficial layer there are numerous goblet cells. The tunica propria is composed of fibrous connective tissue containing many clefts occupied by leucocytes. Occasional nodules of lymphoid tissue are scattered through this. The mucous membrane contains numerous racemose glands which open on the free surface by funnel like pits. They are mixed glands, some of which secrete serous fluids, some mucous products. The veins of the mucosa are so wide and plentiful that the layer in which they run is practically cavernous tissue. The mucous membrane of the accessory spaces of the nasal fossa, the frontal, the maxillary and the sphenoidal sinuses and the ethmoidal cells, closely resembles that of the respiratory region. These tracts are distinguished chiefly by the marked reduction in the thickness of the mucous membrane of the sinuses (.02mm). The meagre supply of glands is another distinguishing feature. (Piersol, Normal Histology, p. 402.)

Arteries.—The arteries spring from the spheno-palatine artery and enter the sinus by its duct. The anterior ethmoidal artery furnishes a few small branches which enter sometimes the posterior face, sometimes the inferior face.

Veins.—The veins make a compact network in the mucous membrane. They are especially developed on the external wall. They are in direct communication with the orbit. There is a second venous connection between the sinus and the orbit through the ethmoidal veins. These pierce the posterior and the inferior wall. The veins of the posterior or the cranial wall come into relationship with the superior longitudinal sinus or better with the vein of the foramen cæcum. The greater part of the venous blood naturally empties into the spheno-palatine vein, the vein which collects the blood from the mucous membrane of the turbinates and the regions between.

Lymphatics.—The lymphatics are a part of the lym-

phatic system of the nose. They are, therefore, in relation, the same as the lymphatics of the nose, with the subarachnoid space by means of minute bony canals in the walls of the sinus.

Nerves.—The nerves of the frontal sinus come from the ethmoidal branch of the nasal nerve. The nerves are very numerous and are arranged in a network which makes the mucous membrane very sensitive.

The Form of the Sinus.—There are two distinct varieties of the frontal sinus, the small sinus and the large.

The Small Sinus.—The small sinus corresponds to the rounded excavation which is ordinarily present at the level of the inner angle of the orbit at its fronto orbital part. This excavation is placed behind the posterior border of the ascending process of the superior maxilla and in front of the os planum of the ethmoid and immediately above the lachrymal bone. The small sinus does not rise above a line drawn between the two supra orbital notches, while its anterior border is limited by a line running up from the crest of the lachrymal bone. When the cavity of the sinus is so reduced the anterior wall is very thick.

The Large Sinus.—The large sinus extends upwards nearly through the glabella and outwards over the inner half of the orbit. Normally in the median line its height varies between 20—22 mm (¾in-1in.). This may be increased in very large sinuses to 40-50 mm (1½in-2in.), the sinus reaching even the frontal protuberance.

The Prolongations of the Large Sinus.—The large sinus is very commonly increased in size by sending out two prolongations, one outward over the supra orbital ridge, and one backward over the orbit.

The Outward Prolongation.—Ordinarily the outward limit of the sinus is 25-27mm (1in.)—from the median line, that is the middle of the supra orbital ridge. Occa-

sionally these limits are exceeded, and the sinus extends some 45-50mm (2in.) to the malar bone.

The Backward Prolongation.—The backward prolongation extends backward on the inner side of the sinus to end blindly about 15-10mm ($\frac{3}{8}$in.), from the rim of the orbit. The height of the prolongation is scarcely 2-3mm ($\frac{1}{8}$in.). It comes into direct relation with the anterior ethmoid cells.

Two other small prolongations of the sinus are sometimes found; a prolongation into the crista galli and a prolongation into the root of the nasal bone.

The cavity of the frontal sinus is frequently narrowed by the ethmoid cells mounding into it. The relationship of the ethmoid cells will be discussed in speaking of the floor of the sinus.

The Septum of the Sinus.—Normally the septum of the sinus is a triangular plate of bone placed vertically between the two sinuses. Its anterior border is attached to the posterior face of the sagittal suture, its posterior border corresponds to the ridge of the frontal bone upon which the falx is inserted, while the apex is uppermost and is placed at the junction of the anterior and the posterior wall. The base is joined with the anterior superior edge of the perpendicular plate of the ethmoid bone. Usually the septum is quite thin, but only rarely are there perforations in it. I found but one undoubted perforation in one hundred heads. When the sinus is very small the septum is thick owing to the development in it of spongy tissue. It may measure in such cases from 6-12mm ($\frac{1}{4}$-$\frac{1}{2}$in.).

The deviations of the septum are extremely variable. On account of its surgical bearing, it is important to remember that no matter how much deviated the upper part of the septum may be the base is nearly always in the median line. Naturally, therefore, the sinus should be entered from as low a point as possible.

In the examination of my specimens I have found that the sinus of one side extended across the middle line and encroached on the territory of the opposite sinus in about 5% of the cases. When such a condition is present the wrong sinus might be opened.

Incomplete or Partial Septa.

In addition to the median septum the frontal sinus often has partial septa. These have two seats of predilection. The commonest place is at the summit of the sinus where the anterior and the posterior walls meet, and the second and more dangerous place is the beginning of the backward or orbital prolongation. Rarer and more accessible septa are found on the median septum itself. These are usually roughly vertical.

The septa which occur in the summit of the sinus run parallel with the median septum, so that the pockets which they make empty freely into the lower part of the main sinus. Where, however, they run obliquely, the reverse of this is true. The thickness of these septa scarcely exceeds 1-2mm; that is, they are about as thick as stout paper. Their size depends upon the distance between the two tables of the frontal. Their height varies between 5-10mm (⅛-⅜in.) I found these small septa in about 10% of the cases. In about 1 or 2% of the cases there occurs a very large septum which almost divides the sinus into two parts. At first sight it seems as if the sinus is completely divided, but just at the lower posterior angle of such a partition there is always a communication with the main sinus. One could readily mistake such a partition for the outer wall of the sinus, unless he had this point in mind and passed a probe round the lower end of the partition into the part of the sinus beyond.

The partial septa of the backward prolongation of the sinus are easier to get at than those of the upper part. Usually there are not more than two or three of these. The

greatest number recorded is four. These septa have a tendency to cross the axis of the prologation obliquely, not to run parallel with it. For this reason the pockets which they make drain very poorly.

The study of the relations of the sinus is the study of the sides which compose it. There are three such sides or faces, and the naso-frontal canal which connects the frontal cavity with the nasal cavity.

The Anterior or Frontal Wall.

The anterior wall, when it exists, is subcutaneous in its whole extent. Exploring the sinus through this wall would be most easy if it were not for its thickness and the great variability of the size of the sinus.

Limits of the Anterior wall.

Where the development of the sinus is normal the anterior wall begins below at the naso-frontal suture and ends in the median line half way to the frontal protuberance. On the sides it extends to the mid point of the orbital rim. Taking subjects as they run on the dissecting table, the absence of the anterior wall occurs more frequently than writers usually state.

In two-thirds of the cases the anterior wall of the sinus corresponds with the anterior wall of the skull. In a third of the cases the sinus is of the small variety and is found just within the rim of the orbit at its superior internal angle—just above the lachrymal bone. As a routine, therefore, the first incision into the sinus should be just at the root of the nasal bone.

The average thickness of the anterior wall is 4mm ($\frac{1}{8}$in. +). It may be as little as 1-2mm or as great as 7-12mm (1-32-$\frac{1}{2}$in.).

Structure of the Anterior Wall.—The anterior wall is ordinarily compact as the orbital rim. For the rest of its extent it is made up of spongy tissue, especially at the frontal protuberances. This layer of spongy tissue is what

gives the free bleeding when the sinus is entered from the anterior wall. Owing to many small channels in the bone this wall is in relation with the external circulation. By means of these infection can travel both inward and outward. The anterior wall often ends superiorly in a set of cells which rim it about like a crown. These cells occasionally require opening up after the fashion of the cells of the mastoid process.

The Posterior or Cranial Wall.—The posterior wall corresponds to the cavity of the cranium. This wall is made up of two parts, a vertical part and a horizontal part. The horizontal part is the roof of the backward or orbital prolongation.

Fairly often there is in the neighborhood of the foramen cæcum a bony canal, running to the cranial cavity and emptying into the frontal sinus. Normally there are openings in the internal wall at the level of the frontal protuberances which give passage to veins which finally end in a sub-meningeal vein of considerable size. At this level the dura mater is firmly attached to the bony wall.

The posterior wall is always in relation with the cavity of the sinus, no matter how small this may be. This cannot be said of the anterior wall.

The Floor of the Frontal Sinus.—Anatomically the floor of the frontal sinus is the orbito-nasal wall because it extends over the roof of the nasal fossa and over the inner part of the anterior portion of the orbit. The floor is either funnel shaped or saucer shaped. Very often the nasal part contains a distinct ledge which in transverse section makes the septal wall of the sinus appear as if a step of bone had been added to it. Under this step runs a branch of the anterior ethmoidal artery.

The Orbital Part.—The outer part of the orbital portion is on a slightly higher plane than the inner or the nasal part. It is generally triangular, and measures transversely

15-29mm (⅝-1⅛in.) and anteroposteriorly 10-15mm (⅜-⅝ in.). In a small sinus the floor consists of but little except this orbital part. In such a sinus the floor is found in the superior internal angle of the orbit. The bone which makes it descends like a wedge between the posterior border of the ascending process of the superior maxilla and the os planum of the ethmoid. Below it articulates with the lachrymal bone. Measuring from the lachrymal bone its height is 12-20mm (½-¾in.). Its width is less extensive, varying between 12-15mm (½-⅝in.). The bone is so thin that it has been broken by a scratch. It participates in all fractures at its level which run to the orbital border and is easily wounded by blunt instruments if they strike under the inner rim of the orbit. Fencing accidents have furnished some classical cases. It offers but little resistance to pus.

The Nasal Part of the Floor of the Frontal Sinus.—The nasal part of the floor is that part which is placed in front of the ethmoidal notch. Anteriorly it articulates with the nasal bones and the ascending process of the superior maxilla. Internally it is in relation with the median septum of the sinus and posteriorly with the ethmoid cells. Externally it is continuous without demarcation with the orbital portion of the floor. In the posterior angle of this quadrilateral if it exists, or if it does not, in the posterior part of the floor and the width of half the ethmoidal notch from the median septum, there is an opening which connects the sinus with the middle meatus of the nose. In the smallest sinuses the floor as such ceases to exist and the cavity of the sinus is pushed outward into the superior internal angle of the orbit and appears simply as an anterior ethmoid cell, which developmentally it is.

The under surface of the nasal part corresponds internally with the roof of the nasal fossa and externally rests upon the ethmoid cells at the summit of the middle meatus. The relation of the floor of the frontal sinus to the nasal

vault is very variable. Sometimes the sinus is directly over the roof of the nose; oftener it is not. In sinuses of a median calibre the floor may be reduced to a slit which crosses the nasal fossa only very slightly. As has just been said, in the smallest sinuses, there is no relationship at all. Furthermore, the sinus of the opposite side occasionally crosses the median line and lies over the nasal fossa on both sides. Even in cases where the floor of the sinus occupies 5-8mm of the nasal vault ($\frac{1}{4}$ in.) the bone may be so thick that it can be penetrated only by a drill. These anatomical considerations make the puncture of the floor of the sinus rarely feasible.

The part of the nasal floor which corresponds to the summit of the middle meatus is of more practical interest than that which is over the nasal fossa. The ethmoid cells frequently mound into this and narrow the cavity of the sinus or make drainage by the natural opening into the nose difficult. It is common to have one of these cells attain a considerable size and project into the sinus, especially posteriorly. Such a cell is often called the frontal bulla. When catheterization of the sinus is successful the catheter generally forces its way through some such cell, and it is at the expense of such cells that drainage is obtained in the operation of enlarging the naso-frontal duct from the sinus down into the nose. The sizable openings into the sinus which one occasionally finds are due probably to the breaking down of some such anterior ethmoid cell which was in relation with the floor of the sinus.

The posterior internal angle of the sinus is of the greatest surgical importance. This is the part of the nasal portion which is in front of the ethmoidal notch. It is the dangerous area of the sinus. Nature seems to recognize this because there is often a small rib of bone running diagonally downward and outward which protects this region. The ethmoid plate often sends forward here a finger-like process which

invites accidents. Also, in this posterior internal angle of the sinus, but on the median septum, however, the vein which runs in the foramen cæcum and is the beginning of the superior longitudinal sinus not rarely mounds into the cavity of the sinus. It should be borne in mind in exploring the sinus that it is necessary to keep away from the median septum at least the width of the corresponding half of the ethmoidal notch, and always to do the strong curretting forward and outward, never backward and inward.

The pulley of the superior oblique muscle is in relation with the floor of the sinus. The best guide to this is the notch for the supra orbital nerve. This is one inch from the median line (25mm). The pulley is three-quarters of an inch from the median line (18mm), one-quarter of an inch nearer the median line than the notch and one-quarter of an inch (6mm) below it. In the wet specimen the pulley is flush with the rim of the orbit, but in the cleaned skull the fossa for the pulley or the spine for the pulley, if this replaces the fossa, is an eighth of an inch (3mm) back from the rim. On the cleaned skull the examination of fifty specimens showed that the fossa for the pulley is roughly half way between the top of the lachrymal bone and the supra orbital notch, and that the lower part of the fossa of the pulley is on a line with the highest part of the naso-frontal suture. The attachment for the pulley occupies a space about one-quarter of an inch (6mm) square. In breaking through the roof of the orbit, therefore, one should go back from the anterior wall three-eighths of an inch (1cc) and should be at least one inch (25mm) from the median line. In order to get far enough away from the anterior wall one must keep close to the posterior or cranial wall of the sinus. This is easier to do if the operator stands behind the patient. The pulley is attached to the periosteum of the orbit and readily strips away with it. It is well, therefore, to consider that if the periosteum is dis-

turbed at all within the orbital rim the pulley is disturbed. In opening a sinus of the small type the pulley is always encountered.

As the frontal sinus is simply an enlarged ethmoid cell the rest of the ethmoid cells, especially the anterior ones, have very important relationships with it.

The scheme of the ethmoid region given by Hajek is the simplest and the most practical. The ethmoid labyrinth consists of a certain number of basal lamellæ with intervening spaces or grooves. The lamellæ with the exception of the first, which is incomplete, extend upward to the cribriform plate and outward to the os planum. The first lamella is made by the unciform process of the ethmoid. It is defective above. The second lamella, the lamella of the ethmoid bulla, is complete and runs entirely through the ethmoid labyrinth. The third lamella gives attachment to the middle turbinate, and the fourth to the superior turbinate. In the spaces between these lamellæ the ethmoid cells develop, and in the adult in part obliterate them. The ethmoid cells are best classified according to their method of draining. They are divided, therefore, into the cells of the middle meatus and the cells of the superior meatus, or according to a little looser classification into anterior and posterior ethmoid cells. The anterior cells drain into the middle meatus and the posterior cells into the third meatus. The number of the cells of the middle meatus is from five to seven, and the number of the cells of the third meatus is three to five. One of two ethmoid cells are generally developed in the lamella of the bulla. This results in the formation of a rounded swelling called the ethmoid bulla which projects into the middle meatus.

The Middle Meatus.—The cavity of the middle meatus has the form of a funnel, flattened transversely. The internal wall of this is formed by the middle turbinate and the outer wall by the outer wall of the nasal fossa. The

base of the funnel is below and the apex is placed above and in front. Upon the external or nasal wall there are two ridges, the ridge of the unciform process and the ridge of the ethmoid bulla. Between these two there is a semilunar groove called the groove of the unciform and back of the bulla a groove, which is not always so well marked, the groove of the bulla. The groove of the bulla generally ends blindly. The groove of the unciform often ends blindly also, and often ends in an ethmoid cell. It is very common to have one or two ethmoid cells developed at the summit of this groove. These cells are in direct relation with the floor of the frontal sinus. Very often, as pointed out above, a cell in this position called the frontal bulla attains a considerable size and mounds into the floor of the sinus.

As has just been said the groove of the unciform generally ends blindly. Once in four cases this groove is directly continuous with the drainage canal of the frontal sinus, the naso-frontal duct. Rather rarely the frontal sinus opens into the groove of the bulla. (6 cases in 150.) In a majority of the cases, however (once in every two cases), the frontal sinus opens separately, above and internal to the unciform groove and in direct line with it.

In the lower part of the groove of the unciform the opening of the maxillary antrum is placed and once in four cases in the upper part, the opening of the frontal and front and back of the groove like grapes from a central stem empty the majority of the anterior ethmoid cells. A few of these empty back of the bulla into the posterior part of the middle meatus. Another ethmoid cell, the cell of the agger nasi, occasionally either mounds into the floor of the frontal sinus or projects backward and blocks the groove of the unciform, or as it is called in the older nomenclature, the hiatus semilunaris. In like manner any enlargement of the ethmoid bulla, especially any forward enlargement,

also blocks this groove. The bulla, therefore, holds the key to the drainage of the frontal sinus, the maxillary sinus and most of the anterior ethmoid cells. As the anterior end of the middle turbinate hides the bulla, practically all exploring and all operating for sinus disease begins with the removal of the anterior end of the middle turbinate.

The anterior ethmoid cells of the upper part of the groove of the unciform not only often mound into the floor of the frontal sinus, but often open into it. Further these cells are often connected in a series with each other and with the sinus. Onodi has pointed out recently that the anterior ethmoidal artery occasionally runs in a half canal in one of these cells. When this condition exists and the cell in which this lies is in connection with the sinus there is another way opened up for infection to spread into the cranial cavity. Again not rarely one of the cells of the groove of the bulla enlarges and comes into relation with the sinus. Instances have been found where such a cell has extended forward to the orbital rim. Such an anomaly has given rise to the report of an accessory frontal sinus. There is probably no such thing as an accessory frontal sinus. Anatomically there is no such thing as the absence of the sinus. It is always present as an ethmoid cell.

The Frontal Canal.—The majority of writers describe in connection with the frontal sinus a canal which unites it with the middle meatus of the nose. Poirier makes this 15mm (1-2in.) long in the male and 10mm (3-8in.) in the female. Its form is cylindrical, but it is slightly compressed from side to side. The diameter varies between 2-3mm ($\frac{1}{16}$-$\frac{1}{8}$in.). It can be entered by bougies corresponding to numbers 6, 7, 8, 9, 10, of the French scale.

The canal cannot be isolated from the cul-de-sacs of the ethmoid cells which surround it and make it. When these are atrophied the duct does not exist and the sinus is in direct relation with the middle meatus. When the cells at

the upper part of the middle meatus are well developed they mound into the cavity of the sinus and lengthen the duct. In one half of the cases a probe can be passed from the frontal sinus through its duct down into maxillary antrum. Pus, therefore, can find its way into the antrum in a still greater number of cases.

The Superior Opening of the Naso-Frontal Duct. The upper opening of the canal is placed the width of the corresponding half of the ethmoidal notch from the median septum of the sinus. Its form and dimensions may be those of the canal below or it may differ greatly from this. Ordinarily the opening is roughly oval. When the ethmoid cells mound into the floor of the sinus they modify its shape, but what is of greater importance, they interfere with the entrance of secretion into it. In one case the opening is placed over and behind the frontal bulla, in another behind and below it. Even when there is no projecting ethmoid cell to narrow its size the opening may be simply a transverse slit, or a slit with the axis slanting toward or away from the median septum. Often the opening is at the bottom of a deep pit and guarded by a rib of bone which slants downward and outward from the median septum.

Catheterization of the Naso-Frontal Duct.—A study of the anatomy of the naso-frontal duct shows that it is only by chance that the sinus can be catheterized. The direction of the canal is very oblique from above downwards and from before backward. The opening of the duct is on the average 50mm (2in.) away from the meatus.

In order to catheterize the sinus it is almost always necessary to remove the anterior end of the middle turbinate. Of course the meatus must be cleared of polypi. The procedure of catheterizing the sinus is as follows: The catheter is bent at an angle a little over or a little under 90 degrees, and introduced between the anterior end of the middle turbinate and the ethmoid bulla. It is

then brought forward and rotated outward until it engages in the groove of the unciform. The handle is then depressed and if the naso-frontal duct is continuous with the groove of the unciform, as it is once in four cases, the catheter glides into the sinus and its handle falls backward and lies snugly against the incisor teeth. In case the groove of the unciform ends blindly the catheter brings up in this pocket or in an anterior ethmoid cell. In the majority of cases the catheter breaks through the base of this ethmoid cell and enters the sinus. If this does not happen and one finds himself in an anterior cell the catheter is withdrawn slightly, rotated inward in order to jump the bar at the top of the groove of the unciform and so enter the opening of the sinus which in one half of the cases is above and internal to this and in a direct line with it. In order to pass a probe from the sinus down into the middle meatus and out of the nose the probe should be bent to correspond to half or three-quarters of a circle two inches in diameter.

ARTICLE VIII.

SOME INTERESTING FEATURES CONNECTED WITH THE OUTBREAK OF SMALL POX AT NORTH ADAMS, MASS., IN 1904.

By LYMAN A. JONES, M.D.
OF NORTH ADAMS.

READ JUNE 13, 1905.

SOME INTERESTING FEATURES CONNECTED WITH THE OUTBREAK OF SMALL POX AT NORTH ADAMS, MASS., IN 1904.

For many years New England, and especially our own Commonwealth, has been a manufacturing rather than an agricultural section. For this reason, in most of our towns and cities, there is a large industrial element in the population. To a considerable extent this latter element is composed of foreigners. This is notably true of the cotton industry, in which many French-Canadians are engaged. By reason of nearness to, and ease of communication with their own homes, annual pilgrimages to some shrine, and the varying requirements of the cotton industry itself, there is a constant coming and going among this class of help, which gives them a markedly floating character.

Small pox is of frequent occurrence in lower Canada. Vaccination is not rigidly enforced, and there is not the dread of the disease which exists here. Hence it follows, naturally enough, that from time to time single cases of small pox, or small outbreaks of the disease, occur in our manufacturing towns, but not for many years has there been so large an outbreak as that in North Adams in the summer of 1904.

A young man, travelling in Ohio with Barnum & Bailey's circus, found himself ill early in July, and being uncertain as to the probable duration of his illness, decided to return to his home in North Adams, where he arrived on July 8th. During his journey an eruption developed, and on reaching home he summoned a physician who made a diagnosis of

small pox. The case was reported immediately to the Board of Health. As the city had no small pox hospital, it was decided to let the case remain where it was, on Eagle St. (1), and within a few hours of the time of its arrival in the city, quarantine was established, the carriage in which the patient rode from the station was fumigated, and so far as known any who had been exposed were vaccinated. Later a nurse was secured to assist the mother in caring for her son. The father, who was not at home at the time, remained away. This was the first case.

On August 2d, a young man, driver of a laundry wagon, living on Luther St. (2), near the first case, was sent to the local hospital, suffering with an illness which was said to be typhoid fever. Two days later, on August 4th, he was found to have small pox. This discovery led to the investigation of what proved to be the third case (3), that of a young woman living in the adjoining house on Luther St., which was fatal on the preceding afternoon.

After an unavoidable delay, required for the necessary seizure of the house on Eagle St., where the first case was, the second patient was removed thither. This course was decided upon by the Board because of its conviction that the contagion of small pox was not readily transmitted through the air, and because so long as there were only one or two cases, it did not feel warranted in putting the city to the expense of seizing other property and in establishing another small pox focus.

The quarantine of the hospital, established with the discovery of the case, was maintained for 16 days.

The local public and the newspapers were somewhat aroused by the occurrence of these two new cases, and some criticism developed at keeping them on Eagle St., where many people passed daily, and the suggestion was made that the retention of the first case there, without guards, had been responsible for the development of the others.

This opinion on the part of the public and press was strongly confirmed by the events of August 6th, when four additional cases were reported, one at 5 Potter Place (4), one at 4 Luther St. (5)—being the second in the house—another at 229 Main St. (6), and the fourth, the father of case one, at 163 River St. (7). As subsequently appeared this was the only case that could reasonably be attributed to case one. He had gone to the outside of the house to carry supplies and do errands.

It is interesting to note that case six, at 229 Main St., a drug clerk, was a frequent visitor at 107 Eagle St., and that he had also been a visitor at 4 Luther St., where were subsequently the two cases already mentioned, and in the public eye this was a further evidence of culpable negligence on the part of the Board.

With these further cases and the uncertainty as to whether there would be others, and with the fact that the house first taken lacked room for additional cases, it became needful to establish other hospital quarters. Several locations on the outskirts of the city were suggested, houses in poor repair, without sewer or water connections, and were even strongly insisted upon by city officials and others, but were as strongly opposed by the Board. On Sunday, August 7th, after inspection two new unoccupied houses on Cleaveland Ave., connected with the sewer, provided with hot and cold water, were rented from the Arnold Print Works. There were five or six other similar houses in the same row, all unoccupied except one, offering an unexcelled opportunity for extension later if needed. On the following day the houses were cleaned, and furnished, nurses were provided, and that evening the patients were removed to the hospital.

The second dwelling was taken for the purpose of having a house of detention, where suspected persons might be under observation, and where the remaining members of

any household might be taken while their own home was undergoing fumigation and cleaning.

Out of deference to public opinion when these four cases and others later were discovered, guards were posted, and in most instances they remained, without reference to the patient's removal to the hospital, until the quarantine period had expired.

The eighth case, apparently unassociated in any way with the others, was reported on Pebble Street (8), on August 11th.

Up to August 22d, nothing had been found to bear out the contention of the Board that the earlier case, with the exception of the boy's father already noted, had nothing to do with the causation of the later cases. On this date, however, seven further cases were reported. Of these the ninth, on Phœnix Street (9), was a man who had been in the local hospital under quarantine until two days before, and while there had occupied a bed adjoining the supposed typhoid case.

The remaining cases, the tenth to the fifteenth inclusive, were all in one house on River Street (10–15).

At this time popular opinion was to the effect that the disease was clearly beyond the Board's control. An investigation of these cases, however, gave the first clew as to the facts and showed as follows:

There were living in the house two married sisters and their husbands together with the children. Three in L's and four in G's family. On or about July 13th, L., accompanied by his family, returned from a three-years' stay in Canada, and went at once to his sister-in-law's home in the south part of the city.

A day or two later, the house being small for the combined families, they moved to larger quarters on River Street. Almost immediately L. himself was taken ill, complaining of chilliness and intense headache. On the fol-

lowing day, yielding to the solicitations of his brother-in-law, he went berrying, but was so miserable that when their destination was reached he spent his time lying on the ground. They returned home at two o'clock and he went to bed. He consulted a physician because of an eruption which appeared on the body and to some extent on the palms of his hands and the soles of his feet. It appears that there was some question as to the nature of his illness, but it was finally decided that he did not have the small pox. He gradually improved, and went to work in the Beaver Mill, August 11. Early in August the baby which slept in the bed with himself and his wife became ill, apparently with the same illness as the father's, but because his sickness had been declared harmless no physician was called. Three days later another child became ill, and so it went until six of the seven children were ill when the same physician was again called, and the disease recognized and immediately reported. This was the second source of infection, and inasmuch as the men of the family had continued with their work daily, and gone about as usual, further cases were looked for. The physician who attended these cases was interested in and daily present at the drug store where case six—the drug clerk—was employed.

On August 24th, second and third cases in the same family on Pebble Street (16, 17) were reported, and on the 26th one at 97 Eagle Street (18). This was probably a second case in the family, for shortly before there had been a case of chicken pox so called, the diagnosis of which for a time had been in doubt. Two cases were reported on the 28th, one on Miner Street (19), and one on Center Street (20).

Because of the Miner Street case, which appeared in a new locality, near a section thickly populated with operatives from the mill close at hand, and because there was reason to suppose that the opportunity for infection had

been greater here, a house-to-house vaccination was undertaken for this and some other sections, though a general vaccination had been ordered some time previously.

This led on the following day to the discovery of a case of small pox in a thirteen-tenement house on Rand Street (21), and later the same day there was found across the street a young girl bearing unmistakable marks of recent small pox, though she was then recovered. Inquiry showed that just before the time of her return to North Adams from Canada, three weeks earlier, she was ill, and that at the time of her return she was broken out with a rash. During the three weeks since her return she had been about freely, and the other members of the household had attended to their usual work. On this and the following day or two, cases were reported at 124 Gallup Street (22), Kemp Ave. Ex. (23), near the hospital, at 363 Main Street (24), 13 Reed Street (25), and Folsom Avenue (26).

House-to-house vaccination resulted in the further discovery on Sept. 2d, of three German families on Reed Street (27–40) where there were fourteen cases of small pox. These cases were all mild and with one or two exceptions, which were still desquamating, were entirely recovered. They were such mild cases that no physician had been consulted. From the history obtainable it was evident that the three families had come to this country within two years, and that the first cases appeared more than two months previously. Two of the families had been in North Adams but eight weeks, and the third only one month. Thus they brought the disease with them, continued at their work, and constituted the fourth source of infection.

Sept. 3d, the 41st case, that of a nurse, was reported. The day before, feeling too miserable to continue her work longer at Floral Lane, she returned to her home on Montana Street (41). This case being in a portion of the

city until now entirely free from infection, was the occasion for a renewed panic almost. One family living near left the city so hurriedly that the clothes were left hanging on the line. The fact that the Board's physician lived in the same block was evidence to some that the disease was being carelessly spread by those who had to do with suppressing it.

By this time too the display lines and news articles of the papers had alarmed neighboring towns and they were attempting unwarrantably to stop traffic and passengers from entering their borders. Two members of the Board of Health of an adjoining town being invited to visit North Adams to see for themselves what measures were being taken in regard to small pox, flatly refused, saying there was "too much intercourse as it was, that was just what they were trying to prevent."

This same day another case was reported on Gallup St. (42). On Sept. 5th there was a case in Willow Dell (43). On the 6th the seventh child in the River St. families (44) was reported, as well as another case on Kemp Ave. Ex. (45). On the 11th a case on Center St. (46), second in the family, and one on Granite St. (47) were found. On the 14th there was a second case at 13 Reed St. (48). On the next day a third case appeared in the Center St. family (49). A priest at 228 Main St. (50), who had been exposed to the disease in attending some of his parishioners at the hospital, was reported ill with the small pox on the 16th, as were also two additional cases in the family on Reed St. (51, 52). Another case was found at 324 Union St. (53). No physician had been called. The statement that this case had been concealed by the family proved true on investigation, for the child had been ill about ten days when discovered. The same day the cook at the hospital (54), who said she had had varioloid years before, came down with a mild attack of the disease. On the 18th there was a second case in Willow Dell (55). Three days later

there was a case at 681 Main St. (56), and on the following day there were reported two cases, one at 510 (57) and the other at 479 Union St. (58). The latter case was that of a young woman who was bookkeeper in her father's grocery store, where many of the people of the neighborhood make purchases. This case had been under the care of a physician for some time and was being treated as a case of skin trouble! The cases on River St., the second source of infection, seem to be responsible for this case, and through it, indirectly responsible for many other cases in the neighborhood; since it was learned afterwards that the father of this case was accustomed to call on the River St. families regularly for grocery orders before the nature of their illness was known.

On Sept. 30th there was a third case on Gallup St. (59), and also one on Union St. (60). In October there were only three cases. On the first, one at 466 Union St. (61). One on North St. (62) on the 10th, and the last on the 14th. This was the second in the family at 466 Union St. (63).

That this outbreak became so extensive was due to the fact that at or about the same time, viz., during the month of July, no less than four separate and independent sources of infection entered the city, only one of which was brought to the knowledge of the Board at the time of its arrival.

Another probable factor in the spread of the disease, was that previous to the outbreak there had been many cases of measles and some cases of chicken pox reported. These two diseases are sometimes confounded with small pox. That such may have been the case here, and thus explain some of the cases otherwise unaccounted for, is shown in one instance where a diagnosis of chicken pox was made, when later a case of small pox actually occurred in the same family. In another instance the physician at the time of his first visit said publicly that he had just visited a typical

case of measles, and on the following day reported the case referred to as small pox.

Still another factor in the spread of the disease was the concealed case on Union St., and the disputed case of the young woman in the grocery store on the same street. These, however, should be considered as secondary rather than independent sources.

While the first case appeared on July 8th, and the remaining sources of infection were not discovered until the period between August 22d and September 2d, it is very gratifying to be able to point out that after Oct. 1 only two new cases were reported,— one of these a second case in the same family,— and that on October 31 it was possible to close the hospital permanently.

During the outbreak the hospital was extended so that four houses were in use, two for hospital and two for detention purposes.

The use of the two houses adjoining the hospital for purposes of detention greatly facilitated the prompt suppression of the outbreak, because it made possible the thorough and immediate fumigating and cleaning of the whole house wherever a case occurred. The idea seemed to be new so far as could be learned, and met with marked approval.

The district on the map enclosed in heavy line, comprising the Reed and Rand Sts. section, where the third and fourth sources of infection were found, was placed under rigid quarantine, with watchmen night and day, until the incubation period was over. Those living in the immediate vicinity of the infection were revaccinated several days later, thus following the custom in some places of vaccinating twice on alternate days. During the quarantine period the Board themselves supplied fuel and provisions, and also paid the rents.

The first source of infection was responsible for two cases.

While it is impossible of proof, there is a reasonable probability that twenty-five cases may be traced to the second, and six to the third source of infection, and that to the two combined five further cases may be traced, making a total of thirty-six cases traceable to these two sources.

The fourth source, the German families, seems not to have directly infected others. This was doubtless due to their lack of acquaintance and intimate association with others, owing to their brief residence in the city and their inability to speak English or French.

There remain eleven other cases, some of which group themselves together, where no reasonable connection can be traced.

As to nationality, twenty-eight cases were in French, fourteen in German, twelve in Irish, one in English, and eight in American families.

Vaccination was uncertain in seven cases, in twenty-three there had been no vaccination. There had been previous vaccination in thirty-three cases, though eight of these had not been vaccinated since childhood.

In the five fatal cases, all adults, three had not been vaccinated, one not vaccinated since childhood, and one was uncertain.

Much more of interest in connection with the management of the outbreak and the manner of carrying on the work might be mentioned, but an apology is already due for having occupied more than the time allotted me.

ARTICLE IX.

OCULAR VERTIGO OF INTEREST TO THE GENERAL PRACTITIONER.

By BENJAMIN P. CROFT, M.D.
OF GREENFIELD.

READ JUNE 13, 1905.

OCULAR VERTIGO OF INTEREST TO THE GENERAL PRACTITIONER.

THERE is one point not directly related to the subject selected that I desire to mention before proceeding. For a number of years past a rather strenuous discussion as to the failure of the general practitioner to properly recognize the results of impaired ocular functions in the production of many of the disturbances which come under his observation has been carried on by some of the leading men in my specialty. That all the knowledge pertaining to the relations of the eye and the other organs of the body is desired, and will be readily accepted, and so far as possible utilized by the intelligent practitioner, I feel very sure. But in order to be available, such information should be presented as much from the standpoint of the general practitioner as possible, and it is important that it should be given with the knowledge that the eye is only one of the many organs which he has to consider in reaching conclusions leading up to a diagnosis. And finally that such labor should be in the form of friendly co-operation rather than in the spirit of a man having a larger knowledge.

I confess to a lingering suspicion that all of these factors have not been met in the discussions above referred to, and I interpolate these remarks here because it is eminently desirable that the specialist should have the most friendly co-operation of the general practitioner. That the writer may contribute toward furthering such a desirable end is one of the objects of this paper.

OCULAR VERTIGO.

For several years vertigo as a symptom has been making its appearance in the histories of my cases of refraction, every once in a while standing out with particular prominence, and I had come to take it as a matter of course, as one does headache, blurred vision or the disturbances of digestion, associated with uncorrected defects of vision. But some time ago I read an article in which the writer, though admitting the possibility of vertigo resulting from eye strain, did not consider it either common or important. This was so contrary to my own experience that I not only instituted a careful review of my old histories but I have inquired with more detail into the histories of the patients that have consulted me since, with the result that I am convinced that ocular defects are a common cause of vertigo.

By vertigo I mean that feeling as if external objects whirled around the person affected, or as if he himself had been whirled; in other words, that disturbance of equilibrium commonly known as giddiness, swimming of the head, or dizziness.

Of the vertigo which forms a part of the symptoms of many diseases, such as cerebral affections lacking ocular manifestations, derangements of the digestive functions or of the circulatory system, I will have nothing to say; you are probably more competent to speak of that than I. Nor again do I intend to cover that interesting phase of the subject which is met with in functional and organic diseases of the auditory apparatus, though it is barely possible that aural and ocular vertigo may be closely associated.

Ocular vertigo, as I have seen it, seems to group itself under three heads:

1. That form seen in paralyses of the ocular muscles resulting from an interruption of the nerve supply of the latter.

2. That seen in paretic states of the ocular muscles, or more properly speaking, ocular muscle imbalance.

3. That type accompanying errors of refraction or what is commonly known as the eye strain.

Class 1.

Of these three forms, that accompanying paralyses of one or more of the ocular muscles has without doubt come under your observation, particularly if, as is usually the case, it is associated with that annoying symptom diplopia or double vision, and I think it is safe to say that in such cases the vertigo is an effect of the diplopia. The two eyes, each forming a separate and distinct image upon their respective retinas, compels the patient to endeavor to separate the true from the false. This effort produces confusion, resulting in anything from a slight whirling sensation to pronounced dizziness, causing one to stagger or even to fall over.

To illustrate such a condition, I will cite the history of a young man, twenty-six years of age, and a mechanic by occupation. A few days before he consulted me, he began to notice a doubling of objects and a distressing dizziness, often feeling even nauseated, and he thought that he would be obliged to give up his work if this symptom continued. External examination showed almost a complete ptosis or drooping of the left upper eye-lid, which upon being elevated by the finger, showed the eye-ball markedly diverged or turned outward, and the patient was as unable to adduct or turn the eye-ball toward the nose as he was to raise the upper lid. In addition, the pupil was widely dilated and he could not read the coarsest type, showing that the internal muscles of the eye, as well as the external group supplied by the motor oculi, were involved.

His family history was negative, and though he denied any specific history I could not find any other accountable

cause; so, fearing that his memory had been affected as was his motor oculi, I put him on specific treatment and he made a steady though slow gain upward. But for a long time after he was able to raise his upper lid and move the eye inward past a line drawn through the centre of the orbit, yet upon removing the blinder from the affected eye and taking a few steps, he would commence to reel and feel faint from dizziness, but eventually his ocular symptoms entirely disappeared. This first example of the production of vertigo under class one, is a good illustration of disease acting as the cause and this disease affecting some part of the central nervous system.

One may also have an accident acting as the cause and the nerve being affected along the course of its distribution. This is illustrated by the following:

Mrs. M. W., aged fifty-two, while walking in the woods tripped over a stump of a tree and, falling forward, struck upon the forehead and right eye. The eye-lids rapidly became swollen, and in two hours the eye was closed, owing to the edema. Hot applications and a pressure bandage were used for three days, when she was able to open the eye-lids. She then noticed for the first time that the vision was much reduced, that all objects appeared double and that she was unable to walk, owing to dizziness, unless she kept the affected eye covered. The double vision remained about four weeks but it was two weeks later before the vertigo disappeared. So much for class one.

Class 2.

Under class two, or vertigo caused by lack of proper balance of the external eye muscles, I would say that I am not sure that a distinct line of demarkation can be drawn between the results of muscular unbalance and that produced by uncomplicated eye strain resulting from an error of refraction, for as yet I have not seen a case of pure muscular unbalance unassociated with some visual defect.

But that faulty eye muscles may be the predominant factor in the production of the vertigo will appear in the further discussion of the class now under consideration.

I have had occasion to use the expression "muscular imbalance" several times, and it may be pertinent for me to explain just what I mean by that phrase. Perhaps I can best make it clear by telling you what I understand to be normal ocular muscular balance. Some one has likened the external eye muscles to a harness, and the eye balls may be said to be swung and held taut in the orbits by this delicate muscular equipment, the four recti muscles tending to draw the globe backward in the orbit and this action being opposed by the tendency of the two obliques to pull it forward. The successful accomplishment of this act presupposes a slight contraction on the part of all the muscles engaged. These muscles, when properly acting, produce a co-ordination of the visual axes, so that when the eyes are directed toward an object these axes will meet exactly in that object, and thus the eyes are said to be balanced.

Of course the process of accurate and comfortable focusing in more complex than this, but the harmonious action of all these muscles is fundamental, and it requires no stretch of the imagination to realize that a constant regular flow of nerve force is also essential.

Now this muscle balance may, from a number of causes of which this paper does not treat, be perverted, and we have muscular imbalance, for if a single muscle or a group of them fails to act conjointly with its fellow or fellows, we have one globe out of parallel, the extent varying from that only possible to measure by delicate instruments to the form discussed under class one of this paper.

Among the many manifestations of this friction resulting from a lack of proper muscle balance is the vertigo of which I am speaking, and its differentiation from classes one and three can be best illustrated by the study of the following history.

A young man consulted me five years ago with the regulation symptoms of eye-strain, but including vertigo, the latter being particularly marked upon going up or down stairs or in other ways changing his elevation. An examination of the eyes showed a small amount of long sight, a great deal of astigmatism, and to all external appearances he had normal muscular balance; but, upon making the regular muscle tests, he was shown to have a marked tendency to abnormal convergence, both when looking at distant objects and at the near point; that is, he could not fix on an object for an instant with both eyes—this effort caused fatigue of a certain group of ocular muscles, the latter would relax, and their antagonists draw the eyes inward. By considerable effort he would balance things again, and thus he see-sawed thousands of times daily.

I first corrected his refractive error, as it was impossible to say at the time which was the predominant cause of his symptoms. I advised his return within a specified time, but, not getting the relief he had hoped, he consulted some one else, who prescribed prisms, which for a time gave him partial relief from his severe headache and annoying vertigo. This cessation of his symptons was only temporary, however, and in due time he returned to me and submitted to an operation on his eye muscle, and he has had a large measure of relief ever since.

This case shows vertigo the result of muscular imbalance not dependent, at least to any great extent, upon the associated eye strain.

Class 3.

In the third class of cases, or vertigo associated with errors of refraction, we find the symptom not at all uncommon, and though I have case records of its occurrence during all periods of life, the most typical examples are seen in those patients who have for years been able to ignore an uncorrected focal error of vision, until the begin-

ning of presbyopia puts an additional burden upon the ocular functions, rendering further comfortable vision, without artificial aid, impossible.

If time permitted one would be tempted to cite cases illustrating vertigo due to refractive errors and occurring during the different periods of life, but I think I will offer you more practical help if I confine myself to those examples occurring about, and probably dependent upon the development of presbyopia.

I have selected three cases, in all of which the vertigo was the most annoying symptom, and they had all faithfully coöperated with their family physician, in the endeavor to locate the cause of the difficulty.

Number one was a merchant forty-six years of age, first seen about two years ago. He had worn glasses for close work for the previous four or five years, always going to a jeweller when in need of anything in that line. For the past three years he had had some headache and eye-pain, which he had steadily ignored, but of late he has had fainting attacks preceded by dizziness. These so-called "spells" are brought on by a hard day's work or if he looks intently at rapidly moving objects, and when he stoops over and quickly assumes the upright posture. He was thoroughly discouraged, because for the past year he had taken different kinds of drugs prescribed by his family physician, and instead of the attacks lessening they had steadily increased, both in severity and frequency. He had about decided to sell his business because his failure to get relief had convinced him that he was developing some brain disease. He came to me simply because a friend with similar symptoms had been relieved by the use of glasses. He was frank to say that he was extremely skeptical as to the influence of the eyes in producing any such trouble. The usual visual tests were made and he was found to be able to read the regular size test types at the normal distance and, with the proper strength glass, was given acute vision at a comfortable reading distance. He also had normal muscular balance. In view of his history, however, one was warranted in suspecting that his ciliary muscles were con-

cealing a focal defect and I advised a complete rest of the eyes from close work for ten days. At the expiration of this period of time he returned, and I paralyzed his ciliary muscles and was gratified to find a small amount of far-sighted astigmatism in the right eye, and a like amount, but of the near-sighted variety, in the left, and the axes of both oblique instead of horizontal or vertical, as is by far the more common.

I neutralized this error by the proper glasses to be worn constantly, gave him an additional pair for close work, and at the end of three months he wrote me that "The glasses helped me a sight." I have seen him twice to change his distant glasses, and in the twenty months since the second examination he has not had an attack of vertigo, and has been practically free from his other ocular symptoms.

Number two was a man forty-eight years of age, and a worker on fine mechanic tools. He is constantly using a gauge scaled to one one-hundredth of an inch. I first saw him in November, 1902, when he told me that for five years previously he had had intermittent attacks of severe pain in the back of the head, preceded or followed by mild attacks of vertigo. For a year before the attacks of dizziness had been growing more frequent and severe until he finally gave up his shop work and secured employment out of doors. At once his "dizzy habit" disappeared, but not associating this relief with non-use of his eyes, after several months he decided to return to his trade. Immediately the old symptoms returned, and as before he held regular conferences with his family physician, but without relief.

Some one finally suggested the possibility of the eyes being at fault, and he came to me, as an oculist, not with much hope but because he desired to eliminate the eyes as a possible factor. He declared that his distant vision was as acute as ever or as it need be, and that his impaired vision for near work was no more than would be expected at his age. Both of these statements I found to be practically true as far as acuteness of vision was concerned, and there was no lack of coördination of his ocular muscles, but I felt justified in treating him as I had the patient above described as number one.

Under paralysis of his ciliary muscles, and after pro-

longed hunting, I dislodged a full diopter of far-sighted astigmatism in both eyes, with the axes oblique as in the other case. Just as soon as this defect was corrected by constantly wearing his glasses and compensation for his presbyopia made for near work, he commenced to have freedom from his symptoms. He reports to me periodically and for over two years he has been entirely free from vertigo, and only has a headache when he commits some indiscretion in eating. He has followed his trade constantly since beginning to use the glasses first prescribed.

Number three was a woman between the ages of thirty-five and forty. I first saw her about two years ago. For seven years previously she had had several attacks of vertigo. These attacks would come on suddenly, and were associated with blindness or blurring of vision. During the past year they have not only been very annoying but even dangerous, and because of them she was prevented from doing many of the necessary details of housekeeping. She had to exercise great care in going up and down stairs; to stand on a chair or step-ladder without falling was impossible. She was very despondent, for she had not had any relief from drugs. She was extremely nervous and irritable, both of which, combined with the attacks of dizziness, she felt were unmistakable evidence of an approaching brain malady.

She had never had any visual defect, and is reluctant to submit to an ocular examination, but does so upon the earnest solicitation of her husband.

Examination of the visual acuity revealed the fact of lowered vision in the right eye, normal vision in the left eye, a large amount of far-sighted astigmatism in both eyes, with the axes again oblique, and practically normal ocular muscle balance. As she positively refused to submit to an examination under a cycloplegic, I was obliged to content myself with prescribing on the basis of the findings of the first examination. I corrected her astigmatism, ordering the glasses for both distant and near use, and advised her return in three months. I did not see her again for eighteen months, when she reported that she had worn the glasses constantly for two or three months, then, not having had any trouble since first commencing to wear them, she

had discarded them entirely. For two months before the second visit her head had commenced to trouble her again, and as the trouble seemed to be increasing, she desired me to look at her eyes again. I found practically the same amount of astigmatism, but the axes had approached the vertical a full ten degrees in both eyes.

She was recorrected and urged to wear her glasses constantly, which she has done with entire relief from her symptoms. This condition will probably be maintained until the onset of presbyopia upsets her ocular equilibrium.

This case cannot be strictly classed as being influenced by the development of presbyopia, but it is a good illustration of the symptom being dependent upon the refractive error; its relief upon the correction of the latter, and the immunity continuing for several months without the aid of the lenses; and lastly the appearance of the vertigo and its final subsidence upon returning to and constant use of the glasses.

I have had a number of similar cases in women, but I call your attention particularly to the histories of the two men because they not only illustrate the class, but demonstrate the theory that men are as likely to be affected as women, but that they are more prone to ignore the influence of the eyes in producing such symptoms.

In conclusion I would say that I realize that the general practitioner should not be burdened with any unnecessary facts, and I have purposely avoided speaking of anything not germane to the particular subject selected, in order that the main proposition, viz., that vertigo could and often does arise from defective ocular conditions, might not be obscured.

ARTICLE X.

THE MEDICAL TREATMENT OF GASTRIC ULCER AND HYPERCHLORHYDRIA.

By FREDERICK C. SHATTUCK, M.D.
OF BOSTON.

READ JUNE 13, 1905.

THE MEDICAL TREATMENT OF GASTRIC ULCER AND HYPERCHLORHYDRIA.

WITHIN the last few years I have modified my treatment of gastric ulcer. For some fifteen years before that it was my rule to starve every case in which the diagnosis was clear for at least fourteen days if possible. In one case of apparent duodenal ulcer in a man of middle age, seen with Dr. C. E. Stedman, rectal feeding was so well borne that stomach rest was carried on for upwards of thirty days. The symptoms never recurred. The principle is, of course, simply that of rest, peristalsis and acid secretion both ceasing, certainly in great measure, in the empty stomach.

I underwent the treatment myself seven years ago, my fast being limited to thirteen days, as rectal feeding produced so much discomfort that I was allowed to give it up. Several years later more or less constant misery from hyperchlorhydria, without bleeding or any other positive sign of ulceration, drove me again to take advice from my brother, Dr. G. B. Shattuck, and Dr. F. Forchheimer of Cincinnati. The experience gained by following their wise counsel has, I know, been of much service to me, and I think I have been able to make it so to others.

Hyperchlorhydria is very common, greater or less in degree, intermittent, or more or less constant; far more as I believe, an expression of faulty innervation, the result of worry or fatigue, than of mechanical irritation or stimulation of the mucous membrane. Diagnosis does not need the stomach tube.

The temporary relief of symptoms by food alone in the milder cases, the prompt relief even in severe cases which follow sodic bicarbonate in sufficiently large doses, one or two teaspoonfuls, clinch the diagnosis. Gastric flatulence, more or less general depression, sometimes very great, with sensitiveness to cold, are suggestive symptoms. Pyloric spasm, as characteristic as it is uncomfortable or even painful — I have known it to start the sweat like angina — is fortunately not very common. It receives scant or no mention in most of the books, possibly because patients cannot accurately describe it without help. As I have experienced it the sequence of events is as follows:

Usually when comfortable, perhaps asleep, largely irrespective of meal time, an uneasy sensation at the epigastrium suggesting distention of the stomach appears suddenly. This sensation increases in intensity, saliva begins to flow, the face and ears feel flushed, yawning and a feeling approaching nausea are also common. Within one or, at the most, three to five minutes, these symptoms recede again. Wind usually escapes from the stomach upward, and relative, more often absolute, comfort returns. Attacks vary in severity.

Experience with patients leads me to believe that pyloric spasm is more common than mere reading would lead one to expect. I crave your pardon for so far indulging in seeming egotism. It is a safe rule to avoid inflicting one's infirmities on others — "to suffer and be still." But the physician who suffers from a malady ought to learn something from his personal experience and be willing to sacrifice his privacy for the sake of benefiting others. Hyde Salter, Morrill Wyman, Oertel and Ralph are names which occur to me in this connection.

There are few conditions more gratifying to treat than hyperchlorhydria. An alkali, only palliative in the more severe and persistent cases, is practically curative in those

which are more transitory, relieving the distress while the nervous system recovers from its fatigue or otherwise rights itself.

Soda mint tablets, I believe, would be better without the mint, and must be taken by the dozen, save in the mildest degrees. It is therefore better to take plain bicarbonate of soda dissolved in plenty of water.

Magnesia, either in powder form or the so-called milk of magnesia, is excellent if there be constipation. But in the higher degrees of hyperchlorhydria the large quantity of magnesia required as an antacid is apt to act too freely on the bowels.

The diet should largely exclude salt, the source of HCl. Condiments should be absolutely excluded. Milk, pure or in combination, or prepared in various ways, is practically always admissible or even desirable, and I often allow soft boiled, dropped or scrambled eggs unless experience forbids. Meat and even fish are to be temporarily excluded, say for a few weeks, and then permitted only sparingly and tentatively.

Diet and such mode of life as tends to restore a more or less tired nervous system, which is apt to be a causative factor, will generally work a cure without the aid of medicine. In severe cases from one-half to two teaspoonfuls of subnitrate of bismuth before meals, seem of distinct benefit, probably acting as a protective coating to the gastric mucous membrane against the food, and thus diminishing the activity and secretion of the peptic glands. In these large doses the drug is not constipating. The microscope shows that the drug has a crystalline formation, which may account for its failure to constipate.

In cases where there is doubt as to whether hyperchlorhydria is complicated by ulcer, the above treatment is applicable, save perhaps that the diet may be made rather more rigid; and this is true also of cases in which ulcer is

probable, but in which bleeding has never occurred, or at all events has not been recent.

In cases presenting marked and characteristic ulcer pain or coming under treatment just after hemorrhage, it is desirable to give the stomach rest for several days. Morphia is to be given hypodermically in such doses as may be required to allay pain. It may also be given in smaller and repeated doses by mouth absorption either for restlessness or to blunt an appetite which it is not then safe to indulge, if circumstances seem to call for it. The drug also quiets peristaltis and diminishes glandular secretion.

After three or four days of exclusive rectal feeding, milk and lime water, a dram every fifteen minutes can be given by the stomach with daily increase in the amount, lengthening of the interval, or both. In ten to fourteen days milk toast, junket, rice with cream and sugar, baked potato, macaroni, blanc mange and the like may be added and rectal feeding omitted. I believe in these cases abstinence from meat should be prolonged for at least three months. Food containing starchy things, such as unstrained oat meal, seed fruits and the like are also to be excluded, lest they irritate the raw surface. The principle is simply to select such food as demands the least stomach digestion and to avoid irritation as far as may be. Bismuth is desirable until full and ordinary diet has been resumed, say three to six months. It here serves a double purpose, diminishing secretion and also mechanically protecting the raw surface from both food and such gastric juice as is formed.

The applications of the principles which I have tried to set forth above have given results in every way satisfactory to me, and I think also to my patients who have followed them out. A recent case in point may be briefly sketched here. An athletic young man without antecedent dyspepsia had profuse gastric hemorrhage in November, 1904.

About two months later he went to New York, omitted his bismuth, and ate irregularly and too largely, although he observed qualitative restrictions. After a week or so he began to have severe pain in the pyloric region with tenderness, came home suffering greatly in the cars and went to bed. Pain and localized tenderness were then more or less constant, excited and increased by examination, persisting in spite of the simplest nutriment, requiring from one to three and a half grains of morphia a day for relative relief. He was then put on a forty-eight hour fast. At the end of this time the pain ceased and convalescence was uninterrupted. He recently came to my office and stated that he should not know that he has a stomach. I believe the persistence in proper diet and bismuth will prevent recurrence, or at least go far to do so. There is, in my opinion, in certain quarters, far too strong a tendency toward operative interference in gastric ulcer. Hyperchlorhydria is a frequent accompaniment of the lesion, if it is not indeed a causative factor. Will operation restore a proper acidity? Will it tend to prevent recurrence? There are other causative factors which are not easy to understand. Why, for instance, does peptic ulcer near the pylorus prefer young women, especially domestics? Why does peptic ulcer prefer the duodenum of men, ten or more years farther advanced in life? Why is peptic ulcer more common in some places than in others, without any notable variation in the customs and mode of life? Why more common in Boston, for instance, than in Baltimore? Whatever the cause or causes of these facts they are not such as the surgeon's knife can remove along with the ulcer or through gastroenterostomy.

Pyloric stenosis, gastric stasis and dilatation due to the cicatrix of an ulcer are curable only by operation. We know that permanent cure follows purely medical treatment of peptic ulcer in say 50% of the cases. Proof is lacking

that surgery can do better. And I personally can see no reason in the nature of things to lead us to believe that it can do better. That an operation can be done with impunity as far as life is concerned does not warrant its performance. Moderate hemorrhage from an ulcer does well under starvation and morphia. So does profuse hemorrhage, oftentimes. It may be a nice question after large or repeated bleeding whether to put salt solution into a vein or under the skin. The slower the circulation the more likely is clotting to occur at the source of bleeding. We have to balance the risk of death from the blood loss which has already taken place against that from the possible starting up of fresh hemorrhage. So delicate may this balance be that error of judgment must sometimes occur. I cannot see much room for surgery in the treatment of ulcer hemorrhage. Mild cases do well without surgery. A severe case would seem to have a better chance without the additional load of an operation. Cases in which pain is so persistent, and hemorrhage so frequent and recurrent in spite of medical treatment, as to justify operation, on the chances as it were, are, as far as my experience goes, excessively rare. The only case in which I have advised operation for gastric hemorrhage was in a young female domestic. No ulcer was found, the blood coming from the whole mucous membrane. She died.

To sum up, cicatricial contraction at or near the pylorus causing dilatation, and perforation, are the only features of peptic ulcer which seem to me to clearly demand surgical treatment in the present state of knowledge. I therefore prefer that further knowledge on this point be gained on the patients of others rather than on my own.*

* For an excellent analytical study of the Surgery of Gastric Ulcer, see Sears, Boston Med. & Surg. Journal, March 30, 1905.

DISCUSSION.

Dr. E. G. CUTLER, of Boston: I agree perfectly with what Dr. Shattuck has said, except in a few minor particulars as to the question of diet and alimentation; and as applying to what he said, I will read a few words which I have put together on the subject.

In the first place, one of the essentials of the treatment is rest in bed, and the time ought to be from three to four weeks. The patient may be on the lounge the fourth week, the time that he is there being gradually increased daily, if borne well. The patient may walk about the fourth week, but should rest on the lounge after each chief meal for a period of one or two weeks longer, when he may go about as usual, but he must not bend forward at work nor exert pressure on the epigastrium for months.

As to diet, I agree with Dr. Shattuck very nearly. I should say, however, that the first week the diet should be largely milk, beginning with small doses every hour, with water as a beverage, up to two or three pints, the amount of milk and times of taking it being gradually increased until six ounces are taken every two hours. The second week, if nausea and pain or discomfort are gone, some of the cereal preparations, well-cooked wheat, or strained barley or oatmeal, or somatose, may be added to the milk. The third week the white of egg, and finally the whole egg may be added to the milk and cereal. The fourth week boiled chicken, broiled sweetbread, baked potato, and bread and milk are added. After this the ordinary diet can be taken, except that no raw fruits, pastry, salads, or coarse vegetables, and I should add beef, should be taken for months, or perhaps for several years. I believe that broths and soups had better be omitted until the patient is well, as they tend to increase the HCl production. It is my opinion that the majority of patients do better by giving exclusive rectal alimentation the first one or two days. A few years ago I advised giving this alimentation for a longer period. It seems to me, however, that you do all you want in two days, or at the most three days, and when six ounces of milk are being taken at a time, the enemata may be omitted. The enema consists of raw milk,

six ounces, one or two raw eggs and half a drachm of salt. The enema to be given once in six hours, and one cleansing enema a day to be used.

Bismuth, in some cases where the pain is persistent, or tenderness present, is advisable, given as indicated by Dr. Shattuck. Another thing which I find of service is olive oil (advised by Conheim), one-half ounce daily, in the morning, and one to two teaspoonfuls in the middle of the day and the evening, it being very effective in acidity and pain. Time and again I have given that with no other treatment except care in diet, in patients with gastric ulcer, and time after time the symptons have disappeared without further trouble. I also frequently use twenty grains of Carlsbad Sprudel Salts, in a little water before each meal. It seems to me to diminish the acidity.

The milk diet should be kept up as long as the Boas examination of the feces reveals occult hemorrhage (guaiac and aloin tests). I have also obtained good results from iron in some form, beginning at about the end of the third week of routine treatment.

For hemorrhage, it has seemed to me that I got more good following out the treatment of Einhorn, who recommends using a syringeful of adrenalin chloride, 1–2000, twice daily, or giving 15 drops of the same solution by mouth, three times daily. I have used this only two or three times, but it seems to be of distinct value. In cases of great weakness infusions of normal salt solution may be used. Profuse hemorrhage recurring at short intervals, or frequent small hemorrhages, I believe demand surgical interference. I also believe that persistent pain, after taking food, and tenderness of the epigastrium, which does not yield to the treatment of rest and diet in three weeks' time, should be turned over to the surgeon.

In looking over my figures, I find that I have had sixty-five cases of gastric ulcer where the diagnosis seemed to be very certain, and where good records were kept, and of that number sixty-one were of the class that perform arduous labor, and four were not; three were doctors who were in the very best of circumstances, and one was a lady patient who was equally well placed. So far as the nationality was concerned, eighteen were Irish, one German, ten Canadian, twenty-nine born in America, nineteen of whom

were of foreign parentage, three English, two Swedish, two from Scotland, and one not given.

As far as results were concerned, five died; there were twenty-seven who were apparently what you would call well when leaving the hospital; four were improved; two much improved; and two not improved at all. I tried to keep trace of these people by postal cards, etc., but as they belonged to a class that is more or less floating and unstable, this was very unsatisfactory. Some weeks ago I wrote letters to every person on this list, known to be living, and got only six answers. Thirty-five letters were written, the dead-letter office brought back ten of them, six answered, and the others, presumably, did not take the trouble to answer, or else they got lost in some way. One replied by postal card, as follows: "I remained well for two and two-thirds years, then I had one slight attack last December (1904)." Another answered: "I remained well up to March, 1905." (That was when I last heard from that patient, and covered a period of five years.) There was one case of hour-glass stomach, which was operated upon, and the patient remained well up to nineteen months after the operation, and the understanding was that we should be informed if anything happened, and as nothing has been heard since then, the patient is probably well. One case where gastrotomy was performed has remained perfectly well for two years. Another case which was operated upon, I saw about a year ago. This was one of the cases which left the hospital as "unimproved." He said he had more or less bad symptoms for seven months, when he was operated upon at the Carney Hospital by Dr. Munro, on Jan. 7, and on March 13 he was very much improved.

The trouble with all statistics, as far as gastric ulcer is concerned, is this, — that the cases occur amongst a class of people who are rather floating in their residence, and relatively irresponsible, and we cannot carry out what we would like. They do not obey directions very well, unless under observation in the hospital, and are therefore unsatisfactory to treat outside.

Dr. M. P. SMITHWICK, of Boston: In regard to this paper, I looked forward to it with great pleasure and listened

to it with great interest, and as far as local medicinal treatment goes, would give just about what Dr. Shattuck believes in giving. But my belief is that the following statements in regard to hyperchlorhydria, or in regard to hyperacidity, in which hydrochloric acid is increased although associated with other acids, are facts:

First. It is fundamentally a neurosis; it may not be a single neurosis, but a combination of neuroses. Hyperæsthesia is always present and clinically is often mistaken for hyperacidity. I see no reason why a hyperchlorhydric stomach, for instance, with a diminished motility, and perhaps altered blood supply, where for hours fermentation goes on and which is filled with bacteria, should have a normal lining. Different grades of gastric catarrh are often present in hyperchlorhydric cases, but improvement in the former is followed by increase of the latter. Increase or decrease of glandular elements reported, may result from nervous influences as has been suggested for ulcer, the latter being possibly due to a so-called angioneurosis or tropho-neurosis.

Second. Clinically, hyperacidity is often with difficulty distinguished from gastric ulcer, and should suggest its possible presence. In thirteen cases that I have examined while ulceration was active, acidity was increased in two, high in one and always less than average in ten. Successive examinations showed marked variation in acidity, all specimens being obtained one hour after the Ewald test-meal. One of my cases, a trained nurse with an ulcer in the pyloric ring, demonstrated at operation, vomited after one or more meals daily for ten years, never experienced pain until a few months before operation, and never noticed blood in vomitus. Another case, with a large chronic ulcer near the pylorus, demonstrated at operation, had never vomited or experienced pain, except heart-burn. Stasis had been demonstrated by another physician two years before operation, and, judging from the history, had caused symptoms four or five years. The diagnosis was made a year before operation. The pain and tenderness in hyperacidity are usually less localized than in ulcer, and usually the first is eased by food, but the most careful study clinically and the most careful search for blood in gastric contents and feces, may leave us in doubt. Every one of

large experience has had such doubt suddenly cleared up by hemorrhage or perforation. The lesson is to treat doubtful cases as ulcer. Cases of so-called latent ulcer may be rare, but atypical ones are common.

Third. Clinically, hyperacidity usually precedes ulcer, and, indeed, usually succeeds it also.

Fourth. If we were to examine normal people, we would find that there is a decided variation in the acidity from time to time, and if we examine neurasthenics or neurotics, we find still greater variations.

The suggestion I would make for the treatment of these cases is treatment of the underlying neurasthenic or neurotic condition. What is needed is to stop getting exacerbations of acidity, and if we can do this the symptoms usually decrease, and the patient can be changed over from a case of gastric neurosis to an ordinary neurasthenic or neurotic. Then he can live in comparative comfort, and in years he may become a comparatively normal individual.

In regard to the treatment of gastric ulcer, I hesitate to say anything, after those of so much greater experience; but I will say that my experience leads me to advocate at least ten days' rectal alimentation, and when food is given by mouth, to prefer milk pancreatized until decidedly bitter. Liquids should be continued several weeks and resumed as soon as gastric distress returns. Absolute rest three weeks.

In the diagnosis and treatment of gastric ulcer the stomach tube should be used only when necessary. Perforated gastric ulcers or those causing a degree of stenosis of the stomach incompatible with nutrition, belong to the surgeon. Excision of the ulcer is a rational procedure, and is the only one that can ever permanently displace medical treatment. The time may come when medical treatment of chronic ulcer will be persisted in only after exploration has shown excision to be impracticable. Mutilating operations, as pylorectomy and gastro-enterostomy, can never be operations of choice.

I would like to say one thing before I leave the subject, and that is, in the study of a digestive neurosis we should search for the cause. If it can be removed we may hope for a cure. Treatment of the local conditions is often necessary, but is only palliative. And here I want to speak of

the cases of two young neurotic girls with strong maternal instincts. After marriage the first trouble was double gonorrhœal tubes, the next was removal of these tubes, and the next was severe hyperchlorhydria which has resisted all treatment, and which will probably continue to resist treatment for some time to come. Saleratus will not cure such cases as these until there has been sufficient time elapse for them to regain nervous equilibrium and to see a baby without envy, and sufficient time for them to learn that other women have their troubles.

In the majority of cases you will find that there are nervous causes; and, fortunately, they can be removed occasionally. More frequently the sufferer can only be helped to a less depressing outlook.

My last and most earnest word will be in regard to prophylaxis. When the parent brings us a child exhibiting neurotic tendencies we should do more than prescribe symptomatic treatment,—we should strive to convince the parent that proper environment and education are the best antidotes for bad inherited tendencies. If the physician does his duty, the formative years in such cases will be dominated less frequently by family tradition, parental ambition, and chance.

ARTICLE XI.

A CLINICAL AND LABORATORY STUDY OF THE THERAPEUTIC VALUE OF HYDROCHLORIC ACID IN DISEASES OF THE STOMACH.

By RICHARD F. CHASE, M.D.
OF BROOKLINE.

READ JUNE 13, 1905.

A CLINICAL AND LABORATORY STUDY OF THE THERAPEUTIC VALUE OF HYDROCHLORIC ACID IN DISEASES OF THE STOMACH.

THE discovery of Hydrochloric Acid as a constituent of the gastric juice is accredited to Prout, 1824. Probably the use of hydrochloric acid as a therapeutic agent in digestive disorders dates from about this time. It was not, however, until about 1871 that it was learned that this acid is absent or diminished in some gastric conditions. The discovery of this fact, together with the results already obtained from its empirical use seemed to place the therapy of hydrochloric acid upon a very firm foundation.

For some years it was generally believed that all cases of severe dyspepsia would show an absence of this acid in the gastric secretion. Even as late as 1883, Leube expressed the belief that this condition would be met in all cases, except those of nervous origin. With this opinion established, it is evident that a strong impetus was given to the further employment of this agent.

But in 1885 Riegel announced the fact, which was soon confirmed by others, that in many cases of severe gastric disorders the secretions disclosed the presence of the *normal* amount of hydrochloric acid. Startling as this announcement may have proven, it soon became obvious that HCl should be employed *only* in those cases in which an absence or a deficiency of the natural acid existed.

Here rests with the majority of physicians the knowledge of hydrochloric acid therapy in stomach disorders upon

the foundation laid in 1885. Having given you, in brief, the history of the use of HCl up to this time, I can best present that not unimportant portion which remains, by quoting from some of our recent writers upon the subject.

Ewald,[1] Diseases of the Stomach. American translation of the third German edition, of 1893. "Hydrochloric acid is of the greatest importance in the treatment of chronic gastric catarrh, because it not alone replaces the deficiency in the secretion and forms acid albuminates so essential for peptonization, but also because it prevents abnormal fermentation, or lessens it, if already present. I order it in as concentrated watery solution as possible, to be taken three to four times after eating, at fifteen minute intervals (that is, about forty-five to sixty drops after each meal). I have employed this remedy for months at a time and without any bad effects."

Einhorn,[2] of New York, in his second edition of Diseases of the Stomach, 1887, he says, "The medicaments were used to a much greater extent in the treatment of chronic gastric catarrh in olden times, than now-a-days. At present we have learned to pay much more attention to diet, to hygiene, and to the mechanical means of treating the stomach. In some instances, however, the medicaments are also serviceable. Among them, HCl is one that is most frequently employed. The idea of supplementing the deficiency of the gastric juice by this acid, which forms its principal element, is most natural. The best way to administer this acid is to give it in drops, six to twelve of the dilute hydrochloric acid in a glass of water, to be taken three times a day, after meals."

Riegel, Diseases of the Stomach, 1903. "Even if very large doses of hydrochloric acid are given in cases of subacidity, the deficit can hardly be compensated, for we can never give large enough quantities of the acid to cause the appearance of free acid in the stomach contents, in cases in

which free HCl was absent before. Hydrochloric acid for therapeutic purposes has a much more limited field than is usually assumed. Judging from my own experience, I am inclined to attach the greatest importance to its stomachic powers (using it alone, or with the bitter tonics, before meals). Possibly it has some value as a disinfectant. Finally it may be given in order directly to aid digestion, although in the light of our present knowledge we can hardly expect marked results."

Such is the recently expressed opinion of a man who may be considered one of the pioneers in gastro-enterology; a physician who has observed the transitions of hydrochloric acid therapy from the time of its early empirical use down to almost the present day.

Mathieu,[4] in 1894, said, "When stasis and fermentation are present HCl may be given in twenty to forty drop doses after meals.

In 1900 he said[5], "I have of late renounced almost entirely the substitutive method of giving HCl on account of the difficulty of giving sufficient doses to obtain results. Very often I have given very small doses of HCl in cases in which they appeared to excite the motor and secretory functions of the stomach. My object was to increase the appetite without the risk of irritating the stomach."

Klemperer,[6] in 1895, "When acid is lacking, one has to choose between sodium chloride and HCl. The latter should be given during digestion, four drops one hour after meals, then six to eight drops at periods of one-half hour. It is more rational, however, to substitute the sodium salt which gives off the acid in the stomach and stimulates the secretion of the glands."

Linossier[7] said, in 1900, "HCl is no longer used to supply a deficiency, but for some accessory properties more or less well established, as its action upon the appetite, its antiseptic properties, etc."

Hemeter,[8] in his work on Diseases of the Stomach, 1897, advocates the use of large doses of HCl.

In an article, in 1903, he says,[9] "Since Prout's discovery of HCl in the gastric juice, this chemical substance gradually became the panacea for all digestive disorders. From this extreme view we have, in recent years, drifted to the opposite extreme, according to which HCl as a therapeutic remedy is entirely dispensable. He still, however, favors its employment.

Thus, you see, no consensus of opinion prevails, yet a general tendency to limit the use of the acid is shown by the later writers.

In entering into the consideration of this subject, let us first note the objects of HCl administration.

1. HCl * in small doses, five to ten drops, either alone or combined with the bitter tonics, is given usually before meals, as a stomachic.

2. In large doses, fifteen to twenty drops, three or four times after meals, it is used to supply a deficiency of HCl in the gastric secretions. The secondary objects to be attained by such use are: (A) to aid digestion; (B) to overcome or prevent fermentation.

3. It is used in both large and small doses with pepsin, with the object of rendering the stomach contents sufficiently acid to enable the pepsin to act (as it is well known that pepsin is inactive except in a properly acid medium).

4. Some believe that this acid stimulates both gastric secretion and peristalsis, also that it excites pancreatic secretion. The acid may be employed with other objects, of which I am unaware.

Experiments to determine the various influences of HCl in the stomach have been made upon animals, healthy individuals, and upon persons with gastric disorders. Undoubtedly the most practical way to ascertain *certain* effects of

* Acidum Hydrochloricum Dilutum U. S. P.=10% of absolute HCl.

this agent is to administer the acid to individuals in whom its use seems indicated, carefully noting its results by the various means at our command. But certain requisites are necessary to such experiments. One must have subjects whose gastric secretions have shown an absence of free HCl, for a considerable period of time, and such absence must result from organic conditions. The influence of the acid used must not be modified by the use of other drugs or medical means. The subjects must be willing to coöperate with the experimenter, to follow directions *not only* as regards the use of the acid, but also as to the diet, habits, etc. *Only* tests carried out under such conditions and during long periods of observation, can prove of much value. In order to present to you the clinical results obtained from the use of HCl in these cases, and that you may be better able to estimate the value of the laboratory tests, it becomes necessary to give a brief history of each case.

Oct. 9, 1903. CASE I. Female, aged 34, married, does her own housework. Stomach trouble since childhood. Appetite excessive, eats five or six times daily. Pain, almost always, with fasting stomach. Relieved by eating. Eructation and regurgitation, considerable. Nausea and vomiting daily of late.

Physical examination. In June, 1903, weigh 99 lbs. Has never weighed 100 lbs. Hearts, lungs, and urine negative.

Palpation. Negative, except left nephroptosis, first degree.

Inflation and Percussion. Greater curvature of stomach 2½ inches below navel and below left costal border.

Fasting contents. Amount 30 c.c., consisting of food and mucus. Free HCl = 0. Ewald test breakfast, amount 40 c.c., ¾ food, mucus abundant. Free HCl = 0. Total acidity, = 24 per C. Combined HCl = .4 per M. Peptic digestion much delayed.

Diagnosis. Chronic gastritis and moderate mechanical insufficiency.

Patient was treated by proper diet, bi-weekly lavage of

the stomach, and a saline before meals until Jan 15, 1904. All subjective symptoms disappeared. The mucus was reduced to a minimum, the motor power of the stomach became normal, and the weight increased to 109 lbs. The gastric acidity remained practically unchanged.

Jan. 15. I began the use of HCl 15 m. and repeat twice at fifteen minute intervals after meals. No other treatment was employed. In the begining the urine was increased in amount, and the patient thought she felt better, but later decided that she felt as well when not taking the acid. The acid was taken faithfully from Jan. 15 to Feb. 23, thirty-nine days. During this period I made ten examinations of the gastric contents. As a result of these tests I concluded the acid was diminishing the secretion of HCl by the stomach, and consequently discontinued its use, although the patient was not conscious of any injurious influence. Later, lavage with sodium chloride, intra-gastric faradism, etc., failed to cause a return of free HCl.

May 2, 1905. Patient reports no gastric symptoms whatever. The weight has increased to 116 pounds. Total observation of case, 19 months.

Case II. April 1, 1904. Female, 26, married. Does part of own house work. Stomach trouble since fourteen years of age, worse for the past four years. Appetite usually good, oftentimes unsatisfied. Pain, in the mid epigastrium, without regard to meals. Eructation, gaseous, frequent, and without regard to meals. Bowels constipated.

Physical examination. Standard weight 150 lbs., now 118 lbs. Heart, lungs and urine negative. Palpation, negative. Size and position of stomach normal. Fasting contents, none. Ewald test breakfast, after one hour, no contents. Double Ewald test breakfast after one-half hour, amount 30 c.c., $\frac{2}{3}$ crumbs. Free HCl = none. Total acidity 22 per C. Cobined HCl = 4 per M.

Diagnosis. Chronic gastritis and pyloric insufficiency.

The patient was treated from April 1st to July 10th by lavage, diet, etc., with marked improvement in all subjective symptoms, but without any material change in the gastric secretions. There was a loss of weight from 118 to 114½ lbs.

On July 10th I ordered HCl, 20 drops, and repeat once, after each meal. While under close observation the acid was taken 24 days, during which period I made seven examinations of stomach contents, and in all about fourteen examinations.

The patient said the acid produced an agreeable feeling in the stomach. At the close of observation I advised the use of 20 drops HCl after each meal as long as it seemed to do good. At this time the patient went into the country for the summer.

Apr. 21, 1905. Patient reports feeling practically well. Thinks she belches more gas than she should. Still takes a dose of the acid occasionally.

Total observation of case 13 months.

CASE III.—March 4, 1904. Female, 26, single, domestic. Referred to me by a physician, with the statement that there had been for some time a marked deficit of HCl in the secretion, but the subjective symptoms had much improved with treatment. Stomach trouble for 2½ years. Appetite poor. Pain, formerly, in mid-epigastrium, after meals. Eructation and regurgitation after meals. Nausea often, but no vomiting. Bowels constipated, using cascara. Frequent micturition, back-ache, and other symptoms, pointed to pelvic trouble.

Physical examination. Standard weight, 155. Present weight 155 lbs. Heart, lungs and urine negative. Palpation, negative except tenderness over the bladder and in left iliac region. Size and position of stomach normal. Fasting contents, none. Ewald test breakfast, amount 100 c.c. ¾ food, mucus in excess. Free HCl = none. Total acidity = 8 per C. Combined HCl = none.

Diagnosis. Chronic gastritis or Achylia Gastrica and pelvic trouble. The patient had taken HCl previously, had also had lavage, diet, etc. I ordered HCl, 8 drops after meals. It was taken from March 8 to April 27, fifty days; at this time the patient underwent a surgical operation. Later, the patient stated that she felt no better while using the acid.

Nov. 7, 1904. Reports that she now has no stomach trouble. Free HCl absent.

In presenting the results of these experiments, this expla-

nation should be made. It was not the original object of these tests to determine all of the various influences of HCl here shown. Had the purpose been such, the conduct of the work might have been different. On the other hand, some of the results obtained are enhanced in value by the fact that they were not anticipated.

1. The immediate effect of large doses of HCl upon the acidity of gastric contents.

Conditions of tests.

Twenty minims of HCl was given in 90 c.c. (3 oz.) of water two or three times, at 15-minute intervals after an Ewald test breakfast. One hour after the meal was given the gastric contents was withdrawn and the acidity tested. Three such tests were made in Case I, and one test in case III—four tests. Results.—The total acidity of contents was always increased, and in three of the four tests, free HCl was present in appreciable amounts. In one test, it was absent.

2. Effect of larger doses of HCl, given after a larger meal.

Fifteen to 25 minims HCl was given in 90 c.c. of water three times within an hour after the ingestion of a large meat sandwich (bread 70 grams, meat 100 grams) and 300 c.c. water.

Six tests were made in Case I, two tests in Case II, and one test in Case III—nine tests in all.

Results.—60 to 70 minutes after the meal was given the total acidity of the gastric contents was always found increased, but free HCl was *never* present.

To some these results may seem surprising. Yet by those familiar with the normal and abnormal gastric secretions, such results would be expected.

The amount of HCl of the normal gastric juice is about 2.5 grams per 1000 grams secretion. (About forty minims of absolute HCl to each quart of juice.) 2.5 grams or forty minims of absolute HCl equals twenty-five grams

or nearly 1 oz. of 10% HCl. Therefore, estimating the amount of gastric secretion at 1000 c.c. or one quart, after an ordinary meal, it is seen that nearly one oz. of 10% HCl would be required to supply the deficit of this secretion if the natural HCl were absent. Consequently the 75 minims HCl, the largest amount used in these tests, could supply only a small part of the deficiency of HCl which existed in these cases.

From the results of the above tests we learn that it is impracticable to administer enough HCl by mouth to supply the deficits of HCl in the gastric juice ordinarily encountered. Therefore the objects attempted by large doses of the acid are either not at all or but partially attained.

3. Effect of long continued use of large doses of HCl upon gastric secretion.

After observing Case I, about three weeks during the use of 45 drops of HCl three times daily, I suspected that the acidity of the secretion had slightly diminished. The tests made during the following ten days convinced me of it. Feeling that this diminution of acidity resulted from the use of HCl, and nothing else, I discontinued it, believing that it was exercising only a harmful influence, although the patient complained of no unfavorable symptoms.

After the discontinuance of the HCl, the gastric acidity gradually increased, though never quite reaching its original degree.

The acidity curves of Case I, Chart No. 1, well illustrate these variations in gastric acidity.

Case II.—The patient took 40 drops HCl after each meal for 24 days. During close observation of this case, I noticed no marked diminution in the gastric acidity, yet at the end I found that the average acidity after the use of HCl was noticeably less than before its administration.

The comparable test of Table No. 2 show an average total acidity of 26 per C. and an average combined HCl of .466 per M. previous to the use of HCl. During the

use of the acid the average total acidity was 22 per C. and the average combined HCl was .35 per M.

The acidity curves of Case II, Chart No. 2, also make this point plain.

Case III.—The patient took eight drops HCl after each meal for 50 days. The four comparable tests made during the observation of the case, show practically no change in the gastric acidity. The total acidity was always very low, and HCl was always absent.

The observations of Cases I and II show conclusively that during the use of HCl the gastric acidity was diminished. In Case I the reduction of gastric acidity during the use of HCl and its subsequent rise after its discontinuance, shows almost beyond question that it was the HCl that caused the diminution of acidity. Observation of Case II tends to show a similar effect of the acid.

It is my impression that the depressing influence of HCl upon the gastric acidity is not generally recognized. In fact, most writers speak of a contrary effect, a stimulative action, and refer in particular to the experiments of Reichman and Mintz and of Du Mesnil.

Reichman and Mintz,[11] in 1892, observed five patients from two to four weeks, and upon each patient made two to five tests of the gastric secretions, during the use of HCl.

In four of these cases free HCl was absent in the beginning, after the use of HCl free HCl returned in three cases, and in one case it returned temporarily. In another case in which free HCl was present, after the use of this acid there was an increase in the amount.

From these meagre observations they concluded that HCl is a stimulant to gastric secretion.

A sufficient report of their cases is not given to enable one to judge of the causes of the diminished acidity, but it is not improbable that in some of the cases the cause was a functional one, and in such cases a return of free HCl may occur *without any* treatment whatever.

Du Mesnil's[12] experiments were made upon individuals whose gastric secretions were normal. As Riegal very

properly states, Du Mesnil's results show nothing more than might be met in the normal fluctuations of gastric acidity, and even if they might be interpreted as showing that HCl increases the gastric acidity in health, it by no means follows that the same influence would be exercised in organic diseases of the stomach.

In the earlier times of gastro-enterology these observations of Reichman and Mintz, and of Du Mesnil, undoubtedly seemed highly creditable, but today, we know that from such limited observations no very definite deductions can be drawn.

Riegel,[17] in 1889, reported a case in which he used HCl, and different authors have cited this case as a proof of the efficacy of this acid as a stimulant to gastric secretion, but Riegel says, "I, personally, am not inclined to consider it a proof."

Reed's observations, in his text-book, seem to be based mainly on his clinical experience, and a study in particular of some cases which he has reported.[14] He says, "In the condition of deficient secretion of the gastric juice, as in atrophic gastritis, prompt beneficial results are obtained by the use of HCl, five to thirty drops, combined usually with pepsin. The truth is, that the usefulness of HCl consists mainly in its action upon the secreting cells, and probably not to any considerable extent upon its power of supplying the place of absent or deficient gastric juice. My own experience, which is amply supported by that of numerous other careful observers, proves beyond question that HCl does, in many cases, gradually bring up the secretion of the normal acid of the stomach to its proper level when deficient, or even almost absent previously.

The use of pepsin, combined with HCl, has for some time been abandoned by most men familiar with gastric therapeutics. Its inutility is so well recognized by practically all who are acquainted with the subject, that its dis-

cussion seems entirely uncalled for. The support of which he speaks, of "numerous other careful observers, that HCl is a stimulant to gastric secretion," cannot I believe be considered as reliable support.

Reed's cases were more closely observed, and for longer periods than those of Reichman and Mintz, and herein his results are more valuable. His concluding statement, however, furnishes a fair estimate of the value of his observations. He says: "Such fragments of clinical experience do not demonstrate beyond question that HCl stimulates gastric secretion,—yet there is a strong presumption as to this effect."

In estimating the stimulative action of any drug upon gastric secretion in patients, the important role of an unirritating diet and good hygiene must not be overlooked.

Absolute proof of a stimulative action of HCl upon secretion will ever be more difficult to obtain than evidence of a negative, or depressing influence.

Pawlow,[15] who introduced 200 c.c., .1 = .5% HCl solution into dogs' stomachs, failed to observe any influence upon the secretion of HCl by the stomach.

Henne[16] introduced pepsin and 400 c.c., .25% HCl solution into the human stomach and examined the gastric contents thirty to forty minutes later. His results, as to stimulation of secretion by the stomach, were negative.

Wolf[17] gave twenty-five drops HCl after meals, and observed no stimulating effect upon secretions.

Stewart,[18] in 1893, treated three patients with HCl, from three weeks to five months, and free HCl did not return. Later he employed lavage, and electricity in two of these cases, and the HCl returned. He concluded that HCl is no use in establishing the natural secretion.

Jaworski,[19] in 1887, carried out some very extensive experiments with HCl, upon patients. He concluded as follows: "The introduction of 200 c.c., .1 = $.20_N$ HCl so-

lution into the human stomach (in various gastric disorders) had no more effect upon HCl secretion, than distilled water; but, after long use of the acid, HCl secretion was depressed.

In so far as I have been able to learn, from a fairly careful search of the literature, Jaworski is the only investigator who has recognized the depressing influence of HCl upon gastric secretions. It is gratifying, indeed, to be able to confirm his results, and I trust our united results will tend to call attention to the depressing effect of HCl upon secretion, an influence of which most writers seem to be ignorant, or to discredit.

I have dwelt at some length upon the action of HCl on secretion, because it is, I believe, the most important function of this agent. If HCl is a stimulant to gastric secretion, it is a valuable, curative, therapeutic remedy; if it is a depressant to secretion, it is a harmful agent.

4. The effect of large doses of HCl upon peptic digestion. In cases II and III the use of large doses of HCl always accelerated peptic digestion.

In Case II, when no acid was given, the time of digestion exceeded 24 hours, after proper acidulation. When the acid was used, digestion took place in 18 hours, as shown by two tests.

In Case III, when no acid was given, the average time of pepsic digestion was about 11 hours, after proper acidulation of the juice. When the acid was used, digestion occurred in from six to nine hours, after proper acidulation.

In Case I the average time of peptic digestion was about seven hours when no acid was taken. When the acid was given, peptic digestion was delayed from four to eight hours, as shown by four tests. Yet when the acid was given with the lighter meal, and the gastric contents rendered positive to free HCl, digestion was accelerated to the short period of $3\frac{3}{4}$ hours.

These tests show that as a rule, peptic digestion is accelerated by the use of HCl, and this I believe is the prevailing opinion.

Yet the four tests in Case I show that in certain conditions large doses of HCl may, in some manner, retard peptic digestion. This latter effect of the acid was a surprise to me, and I am unable, except by conjecture, to account for this action.

My experiments throw little or no light upon the antifermentive action of HCl or its effect on gastric peristalsis.

We know that when present in sufficient amount HCl inhabits lactic acid fermentation. We also know that sarcinæ ventriculi, under certain conditions, thrive in the presence of the normal or even increased amounts of HCl, and that yeast fermentation also occurs under the same conditions.

Some believe that HCl stimulates gastric peristalsis, but any one who has had much experience in the treatment of gastric diseases knows that we possess *far more* efficient means of stimulating this function of the stomach.

Some writers are inclined to attribute a beneficial influence to HCl through its stimulating effect upon pancreatic secretion. Pawlow[20] has shown conclusively that the introduction of HCl into the fasting stomachs of dogs excites the flow of the pancreatic juice. The stronger the HCl solution, up to a certain limit, the greater the amount of secrecretion. But the administration of small or even large doses of HCl after meals, in cases in which the gastric acidity is much diminished, is quite another proposition. Under such conditions, as we have seen, the acidity of the stomach contents is never increased to any considerable extent. For this reason, probably, pancreatic secretion is not stimulated much more than when no acid is used. Moreover it is a question if there is ever any need of such stimulation.

The sum and substance of the experiments and the consideration of the literature here presented yields but little in favor of the use of large doses of HCl, and some evidence that such employment of the acid may be harmful. On the other hand, most writers advocate the use of small doses of HCl. I believe such use of the acid oftentimes results in the alleviation of certain symptoms, but question if it ever improves any function of the stomach. The strong sentiment which prevails in favor of the use of HCl has arisen mainly from the numerous favorable results obtained from clinical experience. In estimating the value of such experience let it be remembered that the vast majority of gastric disorders are of nervous origin, and that probably ninety per cent. of these cases disclose the presence of the normal or increased amounts of HCl. Yet it is chiefly from *this* class of cases that the favorable results from the empirical use of this agent have been obtained. What, then, is the value of such experience? The method, it must be admitted, is not a correct one by which to ascertain the therapeutic value of HCl, because it is generally conceded that the use of this acid is not indicated in such cases.

Clinical experience in gastric therapeutics to-day is not the same kind of experience as was implied by the term twenty or even fifteen years ago.

Now we possess fairly accurate means of determining the action of drugs upon the various functions of the stomach. When these means are employed, then, and only then, we should, and the most of us are willing to abide by the results obtained.

BIBLIOGRAPHY.

1. Ewald. Diseases of the Stomach, English Translation, 1900.
2. Einhorn. " " " 1897.
3. Riegel. " " " English Translation, 1903.
4. Mathieu. " " " " " 1894.
5. " Bulletin gen. de Therapie, Paris, 1900, CXXXIX.
6. Klemperer. International Clinics, 1895, 5s, II.
7. Linossier. Journal de Practiciens, Paris, 1900, XIV.
8. Hemmeter. Diseases of the Stomach, 1897.
9. " American Medicine, 1905, Vol. I.
10. Reed. Diseases of the Stomach and Intestines. 1904.
11. Reichmann & Mintz. Wien. Klin. Woch. 1892, VI.
12. Du Mesnil. Deut. Med. Woch. 1892, XVIII.
13. Riegel.
14. Reed. Internat. Med. Magazine, 1902, XI.
15. Pawlow. Work of the Digestive Glands.
16. Henne. Zeitchrift für Klin. Med. Band XIX, 1891.
17. Wolf. " " " " "
18. Stewart. Therapeutic Gazette, 1893.
19. Jaworski. Deut. Med. Woch. 1887.
20. Pawlow. Work of the Digestive Glands.

DISCUSSION.

DR. M. P. SMITHWICK, of Boston.—In regard to Dr. Chase's paper, I am inclined to agree with him. My last prescription for hydrochloric acid was over a year ago. Observation of a number of patients through several consecutive months has never shown any stimulation of acid secretion from the use of it. I have not been inclined to pay much attention to any lowering of acid secretion, because most of the patients, being neurotics, were likely to show variations. Let me say right here, however, that although I have not been giving hydrochloric acid for a year, it was discarded simply as a stimulant of acid secretion. Some patients with hypochlohydria consider it very beneficial, especially one with heavily coated tongue, achlohydria and hypomotility, and they continue to use it.

CASE No. 1. TABLE No. 1.

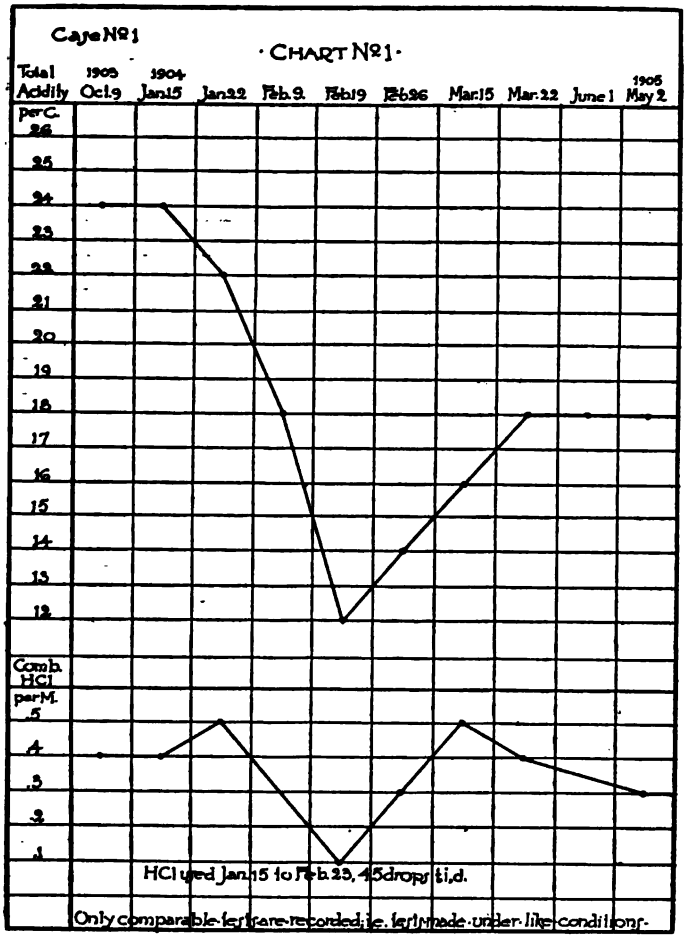
Number.	Date.	Test meal used.	Amount HCl used.	Period after meal.	Amount contents.	Free HCl	Total acidity.	Combined HCl.	Period of peptic digestion.	Rennet.	Rennet gymogen.
	1903		Minims	Minutes	C. C.	per M.	per C.	per M.	Hours	–	–
1	Oct. 9	Ewald	0	60	40	0	24	.4	–	–	–
	1904										
2	Jan. 22	"	0	60	40	0	22	.5	–	–	–
3	Jan. 26	"	40	60	100	0	25	.3	–	–	–
4	Jan. 29	"	40	60	200	1.1	51	.4	3¾	–	–
5	Feb. 5	"	45	60	180	.8	42	–	–	–	–
6	Feb. 9	"	0	60	30	0	18	–	–	–	–
7	Feb. 12	Sandwich	45	70	230	0	40	–	–	–	–
8	Feb. 16	"	50	60	200	0	44	.6	–	–	–
9	Feb. 19	Ewald	0	60	45	0	12	.1	6	–	–
10	Feb. 23	Sandwich	75	75	60	0	40	.5	15	0	–
11	Feb. 26	Ewald	0	60	65	0	14	.3	6¾	+	–
12	Mar. 1	Sandwich	60	60	100	0	36	.5	13	0	–
13	Mar. 3	"	45	60	100	0	20	–	11	0	–
14	Mar. 8	"	0	60	90	0	28	.6	8	+	–
15	Mar. 11	"	60	60	80	0	18	.3	11	0	+
16	Mar. 15	Ewald	0	60	95	0	16	.5	7	0	–
17	Mar. 22	"	0	60	45	0	18	.4	–	–	–
18	Mar. 25	Sandwich	0	60	140	0	20	.4	–	–	–
19	June 1	Ewald	0	30	30	0	18	–	–	–	–
	1905										
20	May 2	"	0	45	65	0	18	.3	8	–	–

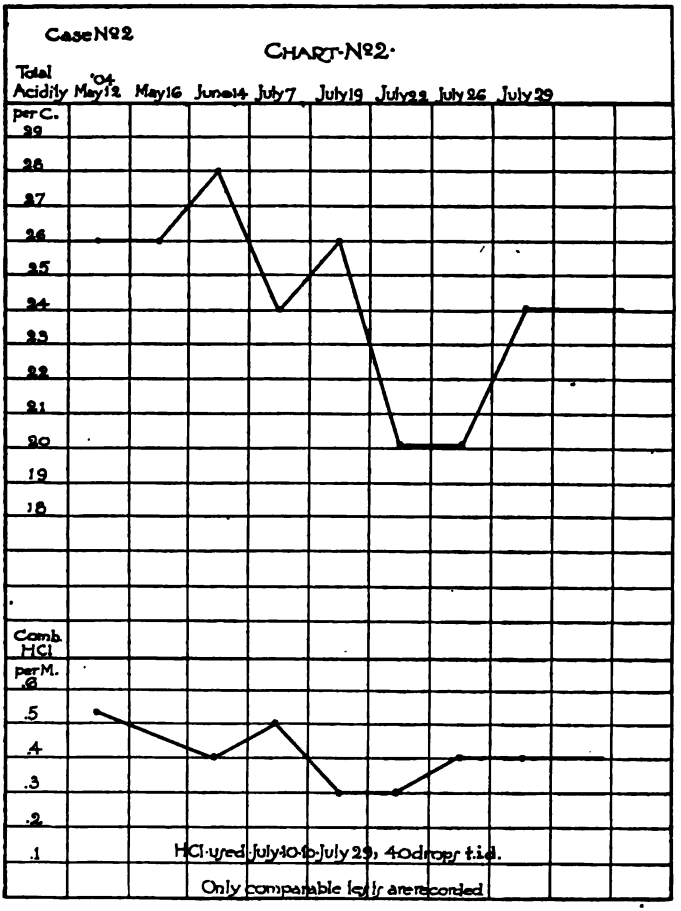
CASE No. 2. TABLE No. 2.

Number.	Date.	Test meal used.	Am't HCl used.	Period after meal.	Amount contents.	Free HCl.	Total acidity.	Combined HCl.	Period of peptic digestion.	Rennet.	Rennet zymogen.	Lactic acid.
	1904		Minims	Minutes	C. C.	per M.	per C.	per M.	Hours.			
1	Apr. 1	Ewald	0	30	20	0	22	.4	0-24	0	-	+
2	Apr. 9	"	0	30	10	0	-	-	-	-	-	-
3	May 12	Sandwich	0	40	120	0	26	.5	0-24	0	-	+
4	May 16	"	0	40	60	0	26	-	0-24	0	-	-
5	June 14	"	0	35	75	0	28	.4	0-24	0	-	+
6	July 7	"	0	30	60	0	24	.5	0-24	0	-	-
7	July 11	"	40	60	120	0	40	.6	18	0	+	0
8	July 15	"	60	60	110	0	40	.5	18	0	+	0
9	July 19	"	0	45	90	0	26	.3	0-24	0	+	+
10	July 22	"	0	40	65	0	20	.3	0-24	0	+	+
11	July 26	"	0	40	350	0	20	.4	-	-	-	-
12	July 29	"	0	50	180	0	22	.4	-	-	-	-
13	Aug. 2	Ewald	0	50	10	0	-	-	-	-	-	-
14	Apr. 2, '05	Sandwich	0	50	5	0	-	-	-	-	-	-

CASE No. 3. TABLE No. 3.

1	1904	Ewald	0	60	100	0	8	0	11	0	+	-
2	Mar. 6	"	0	60	40	0	-	-	13	0	+	-
3	Mar. 8	"	60	60	100	.65	34	0	6	+	+	-
4	Mar. 11	Sandwich	60	60	40	0	-	0	9	+	+	-
5	Mar. 14	Ewald	0	60	60	0	8	-	-	-	-	-
6	Mar. 21	"	0	60	60	0	12	0	10	-	-	+
7	Mar. 24	"	0	60	10	0	-	-	-	-	-	-
8	June 14	"	0	60	40	0	7	-	-	-	-	-
9	Mar. 26, '05	"	0	45	8	0	-	-	-	-	-	-


Case No 1
· CHART No 1 ·
Total Acidity
1903 Oct 9
1904 Jan 15
Jan 22
Feb 9
Feb 19
Feb 26
Mar 15
Mar 22
June 1
1905 May 2
per C.
26
25
24
23
22
21
20
19
18
17
16
15
14
13
12
Comb HCl per M.
5
4
3
2
1
HCl used Jan 15 to Feb 23, 45 drops t.i.d.
Only comparable tests are recorded, i.e. tests made under like conditions.


Case N22
CHART-N22.
Total Acidity per C.
'04
May 12
May 16
June 14
July 7
July 19
July 22
July 26
July 29
29
28
27
26
25
24
23
22
21
20
19
18
Comb. HCl per M.
.5
.4
.3
.2
.1
HCl used July 10-19 July 29, 40 drops t.i.d.
Only comparable levels are recorded

ARTICLE XII.

METHODS IN MEDICINE.

By LEWELLYS F. BARKER, M.D.
OF CHICAGO, ILL.

READ JUNE 13, 1905.

METHODS IN MEDICINE.*

It was a happy inspiration that led the Committee To Procure Scientific Papers to offer as one of the principal attractions of this meeting of The Massachusetts Medical Society a series of demonstrations exhibiting and explaining the various technical procedures pertaining to the practical part of our profession. Nothing could be more interesting or stimulating to the members of a Society of busy practitioners than such an exposition of modern manipulations as, thanks to the sagacity and enterprise of your committee, and the energy and good-will of the demonstrators, has been arranged for; and I can conceive of nothing more likely successfully to entice a group of men from the absorbing and exacting routine in which they are daily immersed, or more helpful and recompensive to them after they have yielded to the seduction, than an opportunity to view the illustrations, here so vividly presented, of the application of all kinds of aids to the art of making accurate observations on patients. While every one of us is doubtless familiar, in a general way, with the great progress which has been making in the modes of examining patients and of gathering data regarding the form and functions of their various organs and tissues, and the constitution of the body-juices, it will come to many of us as a surprise, I feel sure, that there is such a wealth of methods that can be and should be used to help us in our daily work. A great many more helps to

* In the preparation of this address free use has been made of the writings of Claude Bernard, J. Henle, T. H. Huxley, J. Loeb, J. S. Mill, E. Mach, W. Ostwald, K. Pearson, L. Stephen, R. Virchow, C. A. Wunderlich, and W. Wundt.

diagnosis than those actually shown might have been included; many have been unavoidably omitted, but a wise selection has undoubtedly been made, and one sufficiently comprehensive, I think, to convince each of us of his shortcomings. "Knowledge of the condition of a patient is to be drawn out of the patient himself, rather than manufactured without the aid of the senses in the interior of the physician's head." It was to emphasize this fact, Dr. Councilman wrote me, that the committee planned the demostration of methods at this meeting. The only sure way to secure what information is acquirable regarding a patient in addition to what one is told about him, is to examine him in a strictly systematic way, each part in a suitable sequence, with utilization of every aid which will reveal to our senses any deviation from the normal. There is not one of us, I venture to say, who will not return to his practice, in view of what he has here seen, more sensible of his deficiencies and more contrite of his inexactness, than before; each of us will renew his determination to perfect himself in accurate observation and to elevate to the highest level, which his powers and the exigencies of his practice will permit, the standard of his routine. I may speak for myself, at any rate, in this way, and I believe that in voicing this sentiment, I give expression to the thought and feeling of many of you.

Though I have chosen "Methods in Medicine" as a title for my remarks, I shall try to make it clear that, in my opinion, all so-called methods in medicine are but parts of, or aids to, the one great method of acquiring knowledge, viz., the method of science. Science has come to be recognized as universal in its scope; it includes in its legitimate domain the acquisition of truth in every sphere; and whether it deals with the facts of mathematics, the facts of geometry, the facts of physics, the facts of chemistry, the

facts of biology, the facts of psychology or the facts of sociology, it deals with them in one way — in what we call the scientific way. And that way is to collect facts carefully, to arrange them according to their likenesses and sequences, and to interpret them in the simplest manner compatible with verification by healthy minds of our own grade of culture. It is in this way that knowledge in medicine, as in all sciences, has been and is being advanced; it cannot be advanced in any other way.

MEDICINE, AS IT ADVANCES, TENDS TO BECOME MORE SCIENTIFIC.

Biology, far more complicated than physics and chemistry, has, by virtue of the intricacy of its problems, necessarily lagged behind the other sciences, and medicine, the most complex and difficult branch of biology, is still in a primitive stage of evolution as compared with the sciences which deal with the phenomena of the inorganic world. Born of the fear of death, and of the sympathy of man with the sufferings of his fellows, medicine has been compelled from the earliest times to be active as an art before it developed as a science. It is the necessity of action which has always, consciously or unconsciously, led the race, however slowly, to increase its knowing; if a science of medicine is gradually becoming established, it is because in order to do what is right man must first learn to know what is true, because successfully to predict the future he must understand the present and the past. Science began in myth; it passed through dogma; the tendency to-day is to limit its meaning to verifiable truth.

Medicine, like all knowledge, began by chance observation; it has developed through reasoning and the spur to further observation which reasoning has given. But medicine, like all other sciences, and perhaps more than most, has been held back by faulty observation and imperfect reasoning. It has, in turn, been theistic, dogmatic and

metaphysical; it is only very slowly becoming rational, only very gradually scientific. As it progresses, however, it tends more and more to hold to the path of science, for it is learning that in stepping off this road it is invariably led into by-paths which run backward.

The scientific habit of mind is sure to pervade medicine more and more as enlightenment spreads. The enormous advances in theory and practice directly attributable in our time to the application of the method of science to the study of disease and its cure will make us cling to this method as our salvation. We have been taught that there is no short and easy cut to knowledge, such as the ancients sought and hoped for; we are learning to be patient and to be willing to settle the details that are solvable, rather than fruitlessly to attempt to attack the problems of "the whole" that are for us unapproachable; we study, for instance, water as gas, as liquid and as solid rather than the origin of the universe; we inquire as to the characters of the organs and juices in health and disease rather than bother our heads about Vital Force and First Causes. In medicine, as in other departments of knowledge, they who contribute to advance are those who make sure that their facts are well-established, that their classifications of facts are based upon fundamental similarities, that their ideas of the sequence of phenomena conform to actual experience, and that their own conceptions, inferences and interpretations have been so thoroughly criticised by themselves that they will bear the test of the fair criticism of the minds of other healthy and enlightened men.

THE METHOD OF SCIENCE.

The method of science is the only method which may be applied hopefully to the solution of the problems which we meet with as medical men—the problems of diagnosis, of prognosis and of treatment. It is surely important, there-

fore, not only that we should be trained in the use of that method but that we should from time to time take stock, as it were, of our methodological practice to see in how far we are utilizing the scientific method, and in what way, if any, we may do so more perfectly. And such a stock-taking is, I imagine, in part at least, the object of this meeting.

On reviewing the development of any science, especially one which is well constituted, it is easy to recognize three phases through which it has passed:—first, a phase in which facts are established and collected; second, a phase in which facts are compared and arranged according to their mutual relations and connections; and, third, a phase in which by the aid of the imagination these relations and connections are epitomized in the form of brief formulas, or so-called general laws. These three phases are not sharply separable from one another; all three may be met with at once in different parts of the same science. In medicine we are as yet largely in the first and second phases; we are busy collecting facts; we have already done something toward classifying phenomena, toward determining their order in nature, but the science is only slowly reaching the stage of development when the formation of brief laws is feasible. When we remember how greatly thought is economized by the law of gravitation, for instance, or the law of the conservation of mass, or the law of the conservation of energy, or the law of organic evolution, how wide a range of phenomena is summed up in each, we realize the importance of the creation of scientific laws. As these fundmental laws of nature have been found to describe satisfactorily the sequences and relationships for a very large group of phenomena, so in medicine we shall gradually attain to simple statements which will satisfactorily describe succession and relation among more limited ranges of medical facts.

The method of investigation is always the same, no matter what the natural science. The material differs with the individual sciences, the aids to investigation vary, but the mind acts in one and the same way. The groups of sensory impressions which the mind receives and combines with stored sensory impressions of previous conscious states, it projects outside itself; these constitute the only "reality" of physical phenomena possible for us, the only "external world" we know. By association and abstraction these conscious states which are called percepts give rise to other states of consciousness called concepts and from them we draw inferences. These concepts and inferences together with their perceptual antecedents are the facts of science. Science has nothing to do and can have nothing to do with the "beyond" of sense-impressions; if there is anything "behind" them which is the source of them, it is unknowable to us; the "things-in-themselves" of the metaphysicians, scientists know nothing about and need not know. What we do when we acquire knowledge is to collect sense-impressions, store them, combine them, compare them, determine their relationship and order of succession and by an act of creative imagination epitomize descriptions of them into short statements or formulæ. The mind analyzes, synthesizes, abstracts and determines. Induction and deduction are its forms of operation. Observation, reflection, experiment and comparison are their aids. Science would be impossible if the minds of normal human beings of the same culture-grade did not act in much the same way, if the faculties of perception and reflection were not nearly identical among such beings. If the routine of sensory impressions which can be gained were suddenly to cease to be practically the same for one mind that they are for another, or the processes of association and conception to be nearly identical, there could be no agreement among men. One man would create an entirely different external world from that which

another would construct, and every man would be insane to every other man. There could be no general validity for a law; there could be no extensive common knowledge. As the human race has advanced, the capacity for receiving and storing sensory impressions has increased and with it the power of reasoning has steadily developed. Through reasoning man is enabled not only to widen the range of his perceptions but also to summarize them and the concepts derived from them in a way which tremendously economizes his thoughts. He is thus enabled when he receives new sense-impressions to respond more rapidly to them by suitable action. This largely accounts for the superior place he occupies in the struggle for existence.

SCIENTIFIC MEDICINE.

Observing facts, arranging them, and summarizing them in thought-symbols, so as to increase knowledge and economize thinking in order that suitable action may follow most surely and quickly upon impressions of sense is, then, the business of science. Making medical observations, classifying them according to resemblances and succession and epitomizing them in brief statements so as to improve our knowledge of the normal and diseased body in order that we may quickly recognize abnormal phenomena and undertake appropriate treatment is the business of scientific medicine.

Scientific medicine, like all natural science, began with simple observation, with direct sense-impression. Observation alone and unaided, however, cannot bring us very far, and the idea that it can and that medicine is, and must remain, a purely observational science is a grave mistake. Though absurd when one thinks other than naïvely about it, many medical men have fallen into this error; it has been responsible for the serious retardation of medical science. Unaided observation is bound to be very inexact;

through the use of reason, scientific men have learned to experiment and through experiment to invent artificial aids which increase and render more precise the powers of their organs of sense.

The unaided eye can collect only a limited number of sense-impressions regarding the body of healthy and diseased persons, but with the aid of the microscope, the spectroscope, the polariscope, the fluoroscope, the ophthalmoscope, the laryngoscope, the bronchoscope, the cystoscope, and a host of other implements, as well as through the use of chemical reagents and the inoculations of bacteriological culture media, the number of visual impressions can be multiplied more than a thousand-fold. The unaided ear can collect only a few sensory impressions directly from the body, but through the mediation of the stethoscope and by the experimental production of sounds by percussion, the number of auditory-impressions which can be gathered from a patient is augmented. The sense-organs of the physician's skin gave unaided to the earliest doctors and give to doctors today valuable information concerning pressures, pain, coolness, warmth and the like, but that information is minimal in amount and vague in character contrasted with the wealth and exactness of phenomena revealed to us by the instruments which the modern medical man uses as his aids. The sensory impressions, gatherable by the unaided sense-organs, which lead to the conceptions of size, shape, position, weight, movement, time, and rhythm are few and futile in comparison with the sensory data furnished us by the rule, the caliper, the balance, the chronometer, the ophthalmometer, and the graphic recording machines which medical science has applied. The tendency is to pass from the vague to the exact; to replace crude estimation by quantitative measurement expressed in terms of standard units. Medicine has here felt the influence of the physical sciences; it is be-

ginning to be shame-faced when it says "a little" or "a good deal," and is laudably ambitious to express itself in centigrade degrees, in centimetres, grams and seconds, in dynes, ergs, coulombs and Watts.

This tendency to rigid exactness, results in the challenging of every alleged medical fact to make sure that it is a real truth, for medical science has chafed under the particular disrepute into which "medical facts" have somehow fallen, it having before now been sarcastically remarked that "nothing can be less deserving of credit than medical theories, except medical facts." The precision which characterizes the observation of the best modern medical observers would have been impossible had medicine been content to remain an observational science: it has been brought about by reasoning, by the formation of hypotheses and by the subjection of these hypotheses to the control of experiment. The elaborate aids to observation used to-day by the diagnostician are the applied results of physical, chemical and biological experimentation; and further, every time a physician listens to the sounds of the heart through a stethoscope, every time he tests the knee-kick, every time he measures the blood-pressure, every time he makes a Widal test, he performs an experiment in order to extend his observation or make it more accurate. Observation and experiment overlap; to increase his powers of observation, the observer uses implements derived from experimental investigation; these new aids make him observe with greater precision.

An experiment is really an observation provoked at the will of the experimenter; the observation is made then under peculiarly favorable circumstances for the attention of the observer is prepared and his interest is sharpened by the question he is putting to nature. The experimenter commands the phenomenon to appear in circumstances which he has modified; by isolating or varying the circum-

stances he is able gradually to decide as to the relative importance of each, and in many instances he can determine the conditioning circumstance, that is the one which is invariably antecedent to the phenomenon, or what is called its "cause."

Unfortunately in medical science, observers on account of the complexity of the phenomena and their apparent inaccessibility to experiment have been slow in applying the experimental method to medical investigations. As soon as it began to be systematically applied, however, and especially after the recognition of the fact that judicious experimentation on animals permits an actual alteration of the phenomena of life and yields observations which are largely applicable in human physiology, pathology and therapy, medicine began to make progress at an unprecedented speed. Knowledge made so fast that special branches of medicine became established and observers advantageously to themselves and to medical science, began to specialize, to limit their observations and experiments to particular fields. Experimental morphology, experimental physiology, experimental pathology, and experimental therapy are the most helpfully productive sciences in medicine to-day; in them lies our main hope for the progress of medicine in the future.

Next in importance to the help which experiment affords to the scientific method in medicine and really inseparable from it is the aid which comes from comparison. It is this aid which lies at the foundation of various logical methods of procedure, of analysis, of synthesis, of abstraction and of determination. It is the implement which is used in arranging medical facts according to their mutual relations and connections. Phenomena in physiology, pathology, and therapy which resemble one another are graded according to their agreements and differences. As a result of the classifications thus established arise our conceptions

of physiological processes, of disease phenomena, and of the effects of treatment. We all know how the older ideas of the effects of various forms of treatment of pneumonia and of typhoid fever have been rendered ludicrous by the application of the comparative method.

The statistical method, made popular especially by Louis, is an important application of the comparative method; it helped to clear up the vexed questions concerning the continued fevers, and is to-day a prevalent method among medical men. It has its own drawbacks, however, and these too often go unrecognized. To be of value the statistical method must be discriminating; even epidemic diseases vary in intensity in different outbreaks, so that observations made in one epidemic may lead to conclusions very different from those in another. In the use of the statistical method, there are so many pitfalls for the unwary that the taunting remark is sometimes heard that statistics can be made to prove anything in medicine. Unless statistics deal with observations made by one man, at about the same time and upon patients in similar circumstances, the physician who employs them is but too prone to become the victim of many illusions. I am inclined to think that a part of the energy now devoted by hospital physicians to the compiling of statistics could be more profitably employed in the application of the experimental method to the many problems which clamor for solution and are solvable by it. I would not be misunderstood as decrying the legitimate and appropriate use of the statistical method. Far from it; as a part of the comparative method it is a valuable helpmeet to the experimental method in scientific medicine. It is only the imperfect application and exaggerated use of this method to the exclusion or complete subordination of the more important experimental method against which I would register a protest. Comparison is far inferior to experiment, especially for settling questions of cause. A few

experiments will often settle a question of causal relation definitively, where thousands of the most laboriously compiled statistics would throw no light upon conditioning sequence.

The comparative method has been of especial value in the study of symptomatology. The comparison of individual medical cases with all their phenomena led to descriptive or systematic classifications of disease analogous to the classifications of the older systematic zoology and systematic botany. As medicine has become more scientific, the mind has ceased to be satisfied with such descriptive classifications as the clinical symptoms and syndromes represent, and with the "clinical types" set up, and it is ever on the alert to replace them by classifications of a developmental or genetic character. Just as systematic zoology and systematic botany have had to give way to general morphology, so the older nosologies in clinical medicine and surgery are gradually being supplanted by new ones based on etiology and pathogenesis. In the newer classification, generic rather than individual comparison is made. It was experiment that brought about the revolution in classification in zoology and botany; it is the application of the experimental method that is reforming medicine to-day and making generic comparison so much more useful than the old individual comparison. White swelling of the knee, malum coxæ, scrofula, pulmonary consumption, Pott's disease of the spine, tabes mesenterica, renal phthisis, cold abscess, lupus—terms referable to individual comparison—are all joined together in and subordinated to the one term of generic comparison —tuberculosis. Both individual and generic comparison are necessary; the application of the former precedes the application of the latter; but the latter makes more use of induction, is more satisfying to the mind and is sure to come into greater use as medicine becomes more scientific.

Even the term tuberculosis will some day—indeed may now —be regarded as tinctured in many respects with older ontological conceptions of disease, and the time will come, if it be not already here, when we shall have, based on the functional conception of pathology, a still more general grouping of which the function-deviations of tuberculosis will be but special instances. The notion of disease as an entity, of the personification of maladies, will be replaced ultimately by descriptions of the links in the chains of invariable sequences of functional phenomena. In the construction of these higher and more useful thought-symbols, comparison will, however, be less effective than experiment.

Scientific medicine has a great advantage over philosophical or scholastic systems of medicine in that it is independent of personal authority. It demands that its facts and its principles be not contingent upon the decisions of any individual thinker. The successions maintained by it to exist among phenomena and the laws which condense them into brief formulæ must leave no liberty and room for individual fancy. Scientific medicine, using the method of science, contains within itself its own criterion by which its facts, its propositions and its opinions can be tested. A thing is not so any longer because Hippocrates said so, or Galen, or Sydenham. Because Darwin, Virchow or Pasteur held an opinion in their time does not necessarily make that opinion valid in the century in which we live. Knowledge is but relative; it takes new forms as science advances. As the province of experience is enlarged or hitherto disconnected territories become united the older conceptions become inadequate and no longer tenable and must be replaced by new symbols. Verification by experience, not justification by faith, as Huxley was wont to put it, is the touchstone of science. The control of conception by perception, the trial by experience, is the true Ithuriel's spear by means of which we distinguish what is true from what

is false in our ideas of disease and of the treatment of the sick. It would be as wrong for us to-day to accept unchallenged the statements of the great scientists of the 19th century as it was for the followers of Galen to accept his writings as of supreme authority for more than a thousand years. We may admire Virchow, we may venerate the memory of Pasteur, but we shall act best if we do as they did and preserve only those ideas and theories of our predecessors which agree with the collective state of knowledge in our time. There are truths which we can see to day which were invisible to Virchow. There are errors in the opinions of Pasteur which were inherent to his time and which the scientists of this century must try to recognize and destroy. Non-submission to his authority is, in a sense, one of the highest tributes succeeding generations of scientific medical men can pay to the memory of a medical genius.

This devotion to doubt, this consecration of distrust, this systematic questioning of results, is the safe-guard of method in medicine as it is in all sciences. Though the medical man must believe in an invariable order in nature, since all progress in medicine is based upon this belief, still he should always be hesitatingly apprehensive lest he has failed to discover the invariable order in a given case, lest he has confounded a mere coincidence with an inevitable sequence; too often he will find that in with his fact he has unwittingly mixed some purely hypothetical ingredient. The observation that a pneumonia patient recovers after the application of a clay-poultice, that a man with disturbed digestion seems to improve on lithia water, or that a leukæmic spleen diminishes in size under the influence of X-rays, will not justify far-reaching conclusions concerning the treatment of pneumonia, the effect of the alkali metal-ions on a "uric-acid diathesis," or the curability of leukæmia. The scientific medical man,

though often compelled to act without full knowledge, will always be aware of his ignorance, and will ever be tormented by the craving for more precise knowledge. He is forced to act as yet largely empirically, but he will *desire* to act rationally. In his hands the health and the life of a patient will surely be safer, than in the hands of the cocksure healer who knows (*sic*) that he can cure, and is content to be ignorant as to how he does it, or in those of the proud-spirited enthusiast who founds his opinions upon so-called medical tact, upon vague medical intuition or upon some inspired "medical mode of thought" which, in his phantasy, transcends the thinking of science.

Scientific medicine can never be content until it has thoroughly marshalled the facts concerning the body in health and disease, until by observation and experiment and by induction and deduction, it has recognized the relationships of these facts to one another, or until it has formulated general laws which conceptionally symbolize in brief form their regular sequences. A taste of this scientific method and an experience that this method, though dedicated to the principles of "doubt," brings the only "certainty" worthy of the name, should be obtained if possible by every physician early in his career. For the more rapidly the medical profession becomes imbued with the scientific spirit, the more quickly will medicine become physiological, rational, and precise, the sooner will therapy cease to be occult, expectant, nihilistic or merely empirical in order to become judiciously active and definite. The therapy of scientific medicine will not simply avoid hindering the "healing power of Nature"; on the contrary, it will in many cases become able aggressively to interfere with pathological processes and compel the return of normal function.

THE METHOD OF SCIENCE AND MEDICAL PRACTICE.

It has been too much the tendency to separate sharply medical science from the medical art, to contrast knowledge and action as widely divided things in medicine. Too much stress has been laid upon *science* as the mere satisfaction of intellectual curiosity, and upon *art* as mere manipulative skill, largely independent of thought and of systematized knowledge. Convictions concerning the science of medicine and the art of medicine are changing; it is becoming ever clearer that while medicine has two aspects, the so-called theoretical side and the so-called practical side, scientific medicine includes both. On the theoretical side scientific medicine deals, as we have seen, with the search for facts, the arrangement and coördination of facts and the establishment of short generalizations recapitulating the facts and their sequences; on the practical side it deals with the rules and methods for facilitating the performance of the work to be done in making diagnoses and instituting treatments and with the acquisition of skill in the work itself, that is to say in the carrying out of these rules and methods. While it is true that science often makes greatest progress through the efforts of men who avowedly seek truth for truth's sake, independent of any practical application of the truth, and, further, that too great an insistence upon an immediate practical aim in scientific research results very frequently in total sterility of production, an unprejudiced survey of the history of knowledge and action will reveal the fact that the cravings of mankind for brief descriptions and for interpretations, or in other words for *knowledge*, are the impulses which in the long run, through their effects, account for that *action* which has permitted the race to hold its own in the struggle of life; in other words, better *knowing* has always resulted ultimately in better *doing*, the art of making the exertion more fitting has been preceded by progress in the conceptual symboliza-

tion of an ever-expanding body of perceptual experience. The race constantly tends to rid itself, even if slowly, of false observations, reasonings and imaginations, for they are found to impede its progress and not to favor its work; it conserves tenaciously the true observations, conceptions and inferences made by it, for they have contributed to the improvement of its comportment in the past, they are its only guide to safe conduct in the present, and they, together with similar ones to be created by the same method later, form the sole hope for suitable behavior in the future. The two aspects of scientific medicine, the theoretical and the practical, the knowledge side and the art side, ought not to be divorced from one another; if attempts to abrogate the close relation which should exist between them were at any time successful, medical art would be sure to fail to progress, and if the medical art did not advance, it would be bound to deteriorate. Medical science and medical art should live in a state of wedlock. Their conjugal harmony should never be disturbed by lack of appreciation of one for the other.

The objection is often made that medical science being inexact the practitioner cannot be guided by it, but must rely upon his own practical experience in the treatment of the sick. The so-called "practical" man, especially tends to regard theory and practice as incompatible, to deny science and to regard its contributions as useless ballast not needed by the practitioner. I have heard, and doubtless some of you may have heard, physicians of long experience meet the enthusiasms of the medical student regarding progress in scientific medical investigation with the discouraging remark: "The quicker you forget such nonsense the better off you will be. Discard theory; be practical. Do not be a scientist; be a physician." The man who makes such a comment intends to do his young friend a good turn; but he overlooks the fact that scientific medicine includes

the results of the observations and experiments of the most skilled members of the profession for centuries, and does not realize that he is recommending the limited personal experience of one man as its substitute; in opposing theory he forgets that every practitioner, even the crudest empiricist, unconsciously forms hypotheses as to what he sees and uses these as a guide in his treatment, no matter how non-speculative he may think himself to be; he neglects to consider that, instead of the activity of the disciplined mind utilizing the accumulated wisdom of the past, he is advising blind experiment based on instinct and the illogical vagaries of an untrained imagination.

The men who offer such advice look askance at the training given at present by some seven or eight of the better medical schools in this country. A criticism current is that these schools are "making scientists, not practitioners." Let us examine this criticism and see if it be well-worded. If the fact be that these medical schools are turning out graduates who know a great deal about anatomy, physiology, pathological anatomy, physiological chemistry and bacteriology, and nothing or very little about the available methods of diagnosis of disease and the best methods of treating it, I should agree with the critics that the training of the schools was insufficient, but not in their phrasing of the criticism; the criticism should read, "Such schools are educating men in some parts of medical science and not in others, which are equally or even more important for the medical practitioner." But if the fact be that there are medical schools, which are producing, as well as can be done at present, graduates who are versed in the diagnostic and therapeutic knowledge which has thus far been gained either empirically or rationally, and are trained in the application of it, and are, in addition, imbued with the desire to replace empiricism by rationalism, as speedily as possible in every part of the subject; who have learned how to

appreciate evidence, and to form judgments independent of personal bias, who have come to distrust personal authority, and to subject the observations and opinions of themselves and others to the rigid criticism of experience; who have realized that to predict the future accurately and to control phenomena necessitates a foreknowledge of the invariable sequence of events in the domain concerned; in other words, graduates who have learned what the method of science is and how to use it, then the statement that such schools are "making scientists, not practitioners," is mere stultiloquy, for the schools would be making at once scientists and practitioners, *i. e.*, scientific practitioners, rather than merely scientists or merely practitioners. We do not want medical schools to turn out only scientists who cannot be practitioners in the best sense, nor do we desire them to give us practitioners who have no adequate knowledge of science. If medical schools were to try to graduate merely empirical practitioners and not scientific physicians, the inevitable result would be a generation of healers who would be largely charlatans and quacks — a group of self-confident pretenders to medical skill, indolent, ignorant, and incompetent. What has always been needed and is surely most helpful to-day is the scientific practitioner, who recognizes the possibilities on the one hand and the limitations on the other of medical science and the medical art in their present state, who knows that it is necessary to act empirically where reason does not yet guide, but who will abstain from an action which experience has proven to be harmful and will do all that is in his power by observation and by judicious experiment, to advance medical knowledge. We cannot do without the well-trained medical practitioner, who, even if he have but little time to experiment, will be a contributor to medical science, by noting exactly and discriminatingly the phenomena presented by the cases he sees and who will understand which of them can yield profit to science.

The empirical or preliminary period has lasted longer in medical science than in the sciences of inorganic nature, owing partly to the far greater complexity of the problems of medicine, and partly to the antiscientific tendency to rely upon "tact" or "intuition," or the belief that men learn medicine in some way without knowing how, the inclination unduly to exalt the personality of the physician and to diminish the importance of science. Medicine, however, gradually, and more or less unconsciously, entered upon an experimental stage. Physicians early experimented with drugs, but at first they did so very injudiciously, and a Hippocrates was needed to point out the blindness of the early experimentations and to show the value of more thorough observation. By the expectant method Hippocrates proved that many patients suffering from diseases in which cure was attributed to drugs, got well if left to themselves. Mere observation and classification as urged by Hippocrates are not, however, permanently satisfying to either patient or physician, nor should they be. Experiment again had to be resorted to, and gradually experimentation has found profitable directions. It is now clear to scientific medical men that a scientific therapy must be based ultimately upon physiology and that the cultivation and expansion of physiological science is essential if the development of a rational, physiological therapy is to be favored.

While scientific therapy adopts the Hippocratic doctrine of taking advantage of the happy tendencies of nature, it rejects it in as far as it tries to dominate nature in her bad tendencies by actual interferences with processes. It does not wait for the tissues and juices of a child to build an antidote for the poison of the diphtheria bacillus; it hunts for a means of artificially preparing an antidote, secures it, injects it and the child gets well. If the doctrine of expectancy and nihilism had been applied in physics we should

have had no ocean grey-hound, no electric light, no wireless message. We shall miss entirely, or for a long time be deprived of, as great or greater achievements in medical science if we do not push it forward along experimental lines.

Scientific medicine does not undervalue observation or good clinical work; it does not maintain that the laboratories of the auxiliary sciences are the only places whence illumination comes. On the contrary, scientific medicine is more cognizant to-day than ever before that the ideas which stimulate that experimental work which will be fruitful for diagnosis and treatment must come in part, at least, from sick people themselves; it sees a great danger in the almost total separation of the clinic from the experimental laboratories, and is discontent with a condition in which only a very few clinicians are well trained experimenters and only a very few experimenters are well trained clinicians. The scientific physician should have a large bedside experience, should be familiar through personal observation on human beings with the forms of disease upon which he is working, should be able to make accurate diagnoses and true prognoses and have a knowledge of the various remedies which empiricism has recommended, and he should possess real skill in applying them. But he will be more than an observer and more than an empirical therapeutist; he will try to penetrate by the aid of experimentation into the mysteries of disease phenomena in order to untangle them, to understand their proximate causes and to control them. Expectancy and empiricism must for him be supplemented by reflection and experimentation. It is not enough for the scientific physician that mercury and iodide of potassium will cause the changes produced by the virus of syphilis to disappear; he is tormented by the need to know what the virus of syphilis is and to find out how the cure occurs; though he has not yet found out, he will

not be content until he does. Nor is it enough for him that diphtheria has been practically robbed of its terrors by the introduction of the serum treatment; he will be restless until he has isolated from the antitoxic serum the actual antitoxic substance in a pure condition, for the injection of horse's serum into babies, while a great advance on any previous treatment of diphtheria, still reminds him too forcibly of the witch's cauldron.

The scientific physician will not be the victim of any illusion that he himself is to settle experimentally all or even many of the problems which confront him and cry for solution; but he will try to settle at least some of them by experiment himself, and above all he will, under the influence of the scientific spirit which animates him, stimulate his colleagues and pupils to similar work, and support and encourage in every way possible all efforts to make the science of medicine more exact through the application of the experimental method.

It has sometimes been argued that the scientific physician is more at a loss what to do at the bedside of a patient than is the empirical physician. The empiricist is never embarrassed; if one remedy doesn't work, he tries another, and so on. He is often very popular with his patients, for the belief is very common among the laity that medicine has a remedy for every ailment—and that the only difficulty is to lay hands on the right one; a patient will often prefer almost anything to nothing in the way of treatment and the empiricist has always something to recommend. A scientific physiologist or pathologist, who is not also trained as a clinician, would undoubtedly be embarrassed at the bedside of a patient both in making a diagnosis and in advising treatment. The scientific physician must therefore be as well educated on the clinical side as he is in physiology and pathology. Though he sets up as his ideal the establishment of a therapy based on physiology, he will

not when the physiological basis is wanting withhold a remedy which empirical experience has proven useful. In every science parts remain in the empirical state, while other parts become well founded through experiment. In medicine most of our therapeutic measures are as yet empirical and only a few have a physiological or well-grounded experimental basis. Scientific medicine includes empirical medicine as well as experimental medicine, and the scientific physician differs from the empiricist, not in any limitation of resource, but in that he is unwilling to work without continually struggling to pass out of the obscurities of empiricism into light.

An unintentional obstacle to the advance of the scientific method in medicine is to be found I believe in the attitude of some of the representatives of the fundamental sciences of anatomy, physiology and pathology. They sometimes express the opinion that diagnosis, prognosis and therapy are not sciences, but only applications of science, by which they mean that all the clinical man can do is to make applications to his work of the sciences of anatomy, physiology and pathology. This is an old fallacy and it has many analogies. The fallacy lies in the definition of science. The physiologists and anatomists who exclude diagnosis and therapy from the list of sciences would, to be consistent, have to exclude physiology and anatomy also, for physiology is in the same sense largely applied anatomy, applied physics and applied chemestry; similarly chemistry is largely applied physics, and physics, in turn, largely applied mathematics. As a matter of fact all science which deals with concrete phenomena is "applied" science; the only "pure" science is mathematics. This denial of the rank of science to diagnosis and therapy, while physiology and anatomy are accepted as sciences, is an invidious distinction which smacks of the old aristocracy of learning. The sciences which are more precise are not

prone to grant many concessions in the way of recognition to the sciences which are slowly becoming precise. The "pure" chemist shrugs his shoulders when physiological chemistry is mentioned, the physicist used to be shocked to hear engineering called a science, just as the physiologist is to hear pathology and therapeutics referred to as sciences. But science has turned democratic. She welcomes as her own child every domain in which facts are marshalled, arranged and epitomized. Diagnosis has its own group of facts which are gradually being classified according to their complex mutual relations, and expressed in shorthand; so has therapy. Diagnosis and therapy may be subdivisions of physiology if the latter subject be taken in a very broad sense, but then physiology may be regarded also as a branch of physics taken in a very broad sense. The laws of physics do not include, however, all that is known about physiological phenomena, nor do the laws of physiology exhaust everything that can be said about pathology and therapeutics. Pathology and therapeutics are far more complex sciences than physiology and anatomy; the former have new kinds of manifold characters which are not present in the latter. Anatomists and physiologists really wish to see physicians become more scientific; it behooves them, then, to grant that diagnosis and therapy are sciences in the "process of becoming," for by so doing they range themselves on the side of the scientific method in clinical work and not on the side of medical tact, medical intuition, the medical mode of thought, medical inspiration, medical personality, and those other antiscientific, undefinable and non-examinable Divine gifts which the enemies of science find it so convenient to fall back upon.

The method of science, then, is the best method the medical practitioner can use; investigations made in diagnosticating disease and in ordering treatment differ from all other investigations in the materials worked upon, but

not in the method employed. There is only one good method of investigation and it is applicable to all kinds of material. It is the method which makes sure of its facts, which establishes invariable sequences and which summarizes its results in brief statements which economize thought and facilitate prompt and suitable action. It is the method which eliminates personal prejudice so that its results are equally valid for all the normally constituted and adequately disciplined intelligences of the time. Diagnosis and therapy cannot do without this method; it is the sole method of progress.

An attentive examination of the various supports to the scientific method in medicine, demonstrated at this meeting, will reveal the fact that the aids selected are almost entirely concerned with multiplying the possibilities of gaining percepts and of making those percepts definite—in other words, with the increase of the number and accuracy of observations of patients.

It is clear that the committee in charge is of the belief that those of us who attend this meeting can be helped most by being encouraged to collect facts in as great variety, and as exact in character, as is possible through the objective examination of the patient himself.

Other things are of enormous importance, of course, in medical practice,—the arrangement of the facts after they are collected is one, the formation of hypotheses regarding them (to be tested by collecting more facts) is another, the drawing of conclusions based upon comparison with other cases in our experience or in the experience of others, and, where possible, upon experiment, is still another, and, finally, there is the "fitting exertion"—the therapeutic management of the case. These other things, though, are all dependent upon the primary collection of facts in sufficient number and variety, accurately determined. If the data of departure be false or incomplete, the whole course of the subsequent procedure will be vitiated.

The validity of our conceptions and inferences, once the facts are established, will depend upon our powers of reasoning, and our ability to compare and to experiment, but there can be but little hope for valid conception and justifiable inference where the perceptive power is deficient or has been inadequately applied.

It has taken thousands of years to elaborate the methods of clinical examination which are here demonstrated; what strikes one forcibly is the very large proportion of them which have been worked out in the recent past, the direct consequence of the application of the experimental method to clinical science. The results which have been achieved will surely give us increased confidence in man's powers in medicine. As the percepts which can be gained concerning patients grow ever more numerous and complex and our intellectual grasp of physiological function and pathological disturbance becomes continually greater, who will dare to place a limit to the physician's capacity to understand and to master phenomena? I, for one, am unwilling to set one; on the contrary, the history of medicine, especially in the decades just behind us, gives us every reason for assurance that problems of life and mind, of health and disease, of diagnosis and cure, now so dark and so intricate that in many they awaken only despair, will ultimately be solved by scientific physicians. If the past is, as we think it, an index to the future, we need not hesitate to assert with regard to medicine, that, in time, through the further application of the scientific method, *We shall know; we shall be able to predict; we shall gain the power to control.*

ARTICLE XIII.

A Series of Demonstrations Illustrating Methods of Diagnosis in Medicine and Surgery, and also the Causes and Lesions of Disease.

These Demonstrations were organized by Professor William T. Councilman, and the following account thereof has been prepared. Dr. Barker's address was introductory to the demonstrations.

In the past twenty-five years the advance in knowledge of disease has been greater than in any previous century. This increased knowledge has been applied to the problems of disease which are presented to the clinician, and has given greater power in the treatment of disease. Much of the mystery surrounding disease has been removed and medicine is now approaching the status of a science. The increased knowledge has been due to methods of investigation. The methods are based on the fundamental principle that knowledge is derived from observations by means of the senses, and the methods represent extensions of the senses.

It was thought by the authorities of The Massachusetts Medical Society that it would be of advantage to show at this meeting a number of these methods in active operation. The success of these demonstrations has been due to the active work of the men in charge of the different sections. They have involved a great deal of thought and care in preparation.

1.—DIAGNOSTIC METHODS EMPLOYED IN TOXICOLOGY.

The exhibit in toxicology comprised,—the distillation of phosphorus from matches, showing the readiness with

which phosphorus can be procured by a would-be suicide, and the fact that for a few pennies a person can secure enough of this dangerous substance to produce death.

There was shown the deposition of lead upon platinum cones, from urine, which had previously been destroyed with nitric acid, by electrolysis, demonstrating with what readiness the presence of lead in such organic substances as urine can be detected.

There were also shown the different spectra produced by blood which had been acted upon by different gases, such as carbon monoxide, and sulphuretted hydrogen, by such poisons as acetanilid and chlorate of potash, and by strong acids, all of which are of great importance in the diagnosis of such intoxications.

There was shown the extraction of strychnia from solutions of the same mixed with calcium carbonate, by means of the automatic Soxhlet apparatus, which obviates the tiresome process of shaking it out with ether, using the separating funnel.

There was shown arsenical paper and the difference between arsenic and antimony mirrors, and a series of arsenic mirrors produced with the Marsh apparatus, and varying from $\frac{1}{6500}$ to $\frac{1}{65}$ of a grain.

All the ordinary poisons were on exhibition, as well as some of the crystalline precipitates produced by the alkaloidal reagents upon the alkaloids, such as atropine, cocaine and strychnine.

There was shown a ready method of demonstrating the presence of mercury and arsenic in organic mixtures, by which the entire process would be completed in a single test-tube.

There was also shown a rapid method of filtering by which a mixed mass of sulphides or organic matter and metallic poison could be separated when sulphuretted hydrogen had been passed through the mixture, previously destroyed by chlorate of potash and hydrochloric acid, this exhaust filter being run by means of a new electrical pump made by the Boston Vibrator Company.

The demonstration was in charge of Dr. A. E. Austin, assisted by Dr. W. Dacre Walker and Mr. Myron W. Marr.

2.—METHODS OF DIAGNOSIS IN DISEASES OF THE EAR.

The demonstration in Otology consisted in the exhibition of methods of diagnosis by means of objective examination and sound tests, illustrated by patients selected for that purpose; the exhibition of apparatus for purposes of diagnosis, of charts giving the comparative symptomatology of various aural diseases and diagrams and solar enlargements of microscopic sections illustrating pathology.

The exhibit was in charge of Drs. C. J. Blake, Caswell and Wales.

3.—DEMONSTRATION OF METHODS OF DIAGNOSIS IN SURGERY—DRS. J. B. BLAKE AND C. A. PORTER.

Dr. Blake's demonstration consisted of the demonstration of about fifteen patients, a considerable number of X-ray pictures illustrating fine points in surgical diagnosis, and the demonstration of a considerable number of X-ray negatives upon the illuminated screen kindly loaned by Dr. Francis H. Williams, and demonstrated by Mr. Fewkes of the Boston City Hospital. The patients illustrated glanders (with infection and reaction in guinea pig, microscopic slide and culture), intestinal obstruction, exploratory laparotomy, varicocele, hernia, severe compound fracture of legs, fracture of patella, of carpus; malignant disease of rectum, prepatellar bursa, lipoma, abscess, tuberculous ganglion, severe pes valgus, malignant disease of œsophagus and gastrostomy, tuberculous peritonitis with ascites, and several other affections.

Dr. Blake was assisted by Drs. Howe, David Cheever, William Lane, Adams, and G. Blake.

Dr. Porter's demonstration consisted of cases, photographs, pathological material, and some apparatus used in making diagnoses. There were two tables covered with various fresh specimens of pathological material from surgical operations, the object being to demonstrate gross methods of diagnosis—fibroids of the uterus; two knee joints, with amputation of them for septic arthritis; actinomycosis; tumors of the breast, etc.; fluid from spermatocele contrasted with hydrocele. Two patients were demonstrated with a gastrostomy, to illustrate the methods of diagnosis of diseases of the œsophagus, swallowing of string for impermeable stricture; with demonstration of

the various instruments—bougies, sounds, etc., belonging to Dr. S. J. Mixter.

The clinical cases, numbering in all about twenty-five, showed numerous fractures of interest to the general practitioner—old unreduced Colles' fractures, old unreduced dislocation and fracture of the upper end of the humerus, Pott's fracture, one united, one ununited, etc.; result after excision of the tibia for osteomyelitis; case of cured actinomycosis of the pleura and lung; idiopathic gangrene; symptomless hematuria; hydrocele; hernia; tumor of the testis; tuberculosis of the knee; caries of the cervical vertebræ; abscesses of the neck; spontaneous gangrene of hand; and two especially valuable demonstrations of advanced keloid following burns, etc.

About fifty photographs were arranged, with legends attached to each showing some of the commoner as well as rare surgical lesions—various photographs of the two cases of keloid before and after operation; cases of cancer in special parts of the body; contrast photographs of the different varieties of hernia, exstrophy of the bladder, syphilitic diseases of bones; series of cases of carbuncle before and after excision; plastic operations; empyema, with characteristic enlargement of the terminal phalanges; some colored photographs such as mesenteric thrombosis of the intestines, and an interesting series of the special infections —anthrax, actinomycosis, glanders, tetanus, etc., —the special object of these being to illustrate the points which would enable one to make a differential diagnosis, and the results before and after operation.

So far as possible the fractures shown and the cases of bone disease were illustrated either by the X-rays or from prints.

Dr. Porter was assisted by Drs. William C. Quinby, Harry Hartwell, John Little, Channing Simmons, and Charles Butler.*

* "Of my part of the exhibit, it seemed to me that the general practitioners were interested in the rare cases, such as the keloid, to some degree, but especially in demonstrations of the more common lesions, such as fractures, 'house-maid's knee,' and the scrotal tumors. Were such a demonstration to be repeated, I should advise more attention and care being given to an exhaustive showing of all of the common surgical lesions without much regard to the real rarities.

The patients, on the whole, on finding that a great many other men and

4.—METHODS OF DIAGNOSIS IN SKIN DISEASES.

In the section of Dermatology cultures and microscopic preparations of the various forms of vegetable parasites were demonstrated. The affections which these agents produce were illustrated by patients and by numerous drawings and photographs. A number of patients with scabies were shown to illustrate the method of diagnosis and to emphasize the prevalence of this affection at the present time and the difficulty of diagnosis in cleanly people. There were also shown a number of wax models illustrating this affection. Microscopic preparations showing various cutaneous lesions were also exhibited.

The demonstration was in charge of Drs. Bowen, White and Burns.

5.—METHODS OF PHYSICAL DIAGNOSIS IN INTERNAL MEDICINE.

Cases were demonstrated as well as methods of diagnosis. Examples of the following diseases were shown:

Pernicious Anæmia in remission (2 cases);
Leucæmia under X-ray treatment;
Plumbism;
Acromegalia;
Paget's Disease;
Raynaud's Disease;
Mitral Stenosis (4 cases);
Mitral Regurgitation (4 cases);
Aortic Regurgitation (3 cases);
Congenital Heart Lesions (3 cases);
Tuberculous Peritonitis;
Obstructed Vena Cava inferior.

This demonstration was in charge of Dr. Richard C. Cabot, assisted by Drs. N. W. Chandler of Medford, G. S. Hill of Marblehead, F. R. Jouett of Cambridge, J. C. Donoghue of Boston, F. P. Denny of Brookline, F. J.

women were going through the same treatment, and upon payment of their railroad fare, and a small additional fee for their trouble, made no objection at the time or afterwards to their treatment. On the other hand, the absence of sheets and pillow-cases and the inadequacy of protection for the women, from an absence of screens, made the demonstration in the surgical department, as well as in some other rooms, appear to me a little wholesale, and more money, judiciously spent, would change for the better the entire aspect." C. A. P.

Foley of Worcester, J. J. Whoriskey of Cambridge, J. B. Hawes 2d of Boston, Merrick Lincoln of Worcester, W. C. Bailey of Boston.

6.—DEMONSTRATION IN PATHOLOGICAL ANATOMY.

It was thought advisable to combine with this demonstration of methods the demonstration of preparations showing the lesions of the disease.

The demonstration in pathological anatomy consisted of a series of fifty framed bromide enlargements of microphotographs illustrating the pathological histology of smallpox, typhoid fever, cirrhosis of the liver, tuberculosis and certain malignant tumors, and a series of seventy-five paintings of lesions of bones and joints.

Besides this there were a large number of gross specimens preserved in Kaiserling fluid and mounted in gelatin, either in flat rectangular glass cells or under Petri dishes cemented to rectangles of plate glass. The microphotographs in many cases illustrated the finer histological points while the gross specimens showed in natural colors the gross appearances of the same lesion. These were displayed on eight large tables.

The demonstration was in charge of Dr. H. A. Christian, assisted by Drs. W. R. Brinckerhoff, E. H. Nichols, E. E. Southard, E. E. Tyzzer, and S. B. Wolbach.

7.—METHODS IN DIAGNOSIS BY MEANS OF X-RAY.

No especial demonstration of the technique of taking X-ray pictures was given because the different forms of apparatus were shown by the makers in the Exhibition Hall.

The demonstration of "The X-ray in Medical Diagnosis" consisted of charts, plates, prints and skiagraphs, illustrating as much as possible of the following conditions:

1. *Head.* (1) trauma, and (2) abscess of brain. Various pathological conditions in the antra.
2. *Neck.* (*a*) Goitre (cystic type).
   (*b*) Tubercular glands (caseous).
3. *Chest.* (*a*) Lungs.
   (1) Tuberculosis.
   (2) Pneumonia.

(3) Pleurisy with effusion.
(4) Empyema.
(5) Hydrothorax.
(6) Pneumothorax.
(7) Emphysema.

(*b*) Heart.
(1) Displacement.
(2) Transposition.
(3) Hypertrophy.
(4) Dilatation.

(*c*) New Growths.
(*d*) Aneurisms.

4. *Abdomen.*
(*a*) Stomach (1) size; (2) position; (3) tumors.
(*b*) Liver. (1) In children. (2) Subphrenic abscess.
(*c*) Gall Bladder (1) calculi.
(*d*) Kidneys (1) calculi; (2) pus; (3) tumors.
(*e*) Ureters (1) calculi.
(*f*) Bladder (1) calculi.
(*g*) Pregnancy.

5. *Bones.*
(*a*) Osteo-arthritis.
(*b*) Tuberculosis.
(*c*) Osteo-myelitis.
(*d*) Sarcoma.
(*e*) Necrosis.
(*f*) Syphilis.
(*g*) Rachitis.

The demonstration was in charge of Dr. F. B. Granger.

DEMONSTRATION OF X-RAY IN SURGICAL DIAGNOSIS.

Since the essential part of the diagnosis by means of the X-ray is in the interpretation of X-ray pictures, a brief lantern slide demonstration was arranged and repeated at intervals during the morning. Plates which were shown were selected to illustrate the different appearances which are characteristic of different forms of disease affecting the bone, and the points which were characteristic in the X-ray picture of each disease were explained; *e. g.*, Plates were

shown illustrating the fact that portions of the bone cast shadows of an intensity directly proportionate to their atomic weights, and hence only the portions of the bone which normally are partially formed of lime salts will appear in the X-ray picture. Those portions of the bone which are constructed of organic matter, on the other hand, do not appear in the X-ray picture at all. However, since different forms of disease affect the calcified structure of the bone in different ways, one may infer the condition of the soft parts from the manner in which the dense portion of the bone is invaded. To illustrate this point, plates were shown of the more common lesions which affect the bone.

Points like the following were especially dwelt on and illustrated by special cases. Tuberculosis rarely affects the shaft of the bone and nearly always affects the epiphyseal end. Syphilis on the other hand rarely affects the epiphyses, almost always the diaphysis. In the early stages tuberculosis is characterized by loss of bone substance, whereas syphilis nearly always causes hypertrophy of the bone. A blistered appearance on the surface of the bone is characteristic of syphilis. Tubercular sequestra are always small and usually poorly defined, while the sequestra of osteomyelitis are generally large and clearly defined. Bone abscesses caused by tuberculosis are usually small, while those caused by septic processes are large.

In a similar way the appearances characteristic of medullary and periosteal sarcoma, chondroma and osteoma, and hypertrophic and atrophic arthritis, and other forms of bone diseases, were spoken of and illustrated.

This demonstration was in charge of Dr. E. A. Codman.

8.—METHODS OF DIAGNOSIS IN DISEASES OF THE NOSE AND THROAT.

Slides of the usual pathological conditions of the nose and throat were demonstrated, X-ray plates in series were displayed showing their value in locating the frontal sinus and determining its size. There was a second series of plates of foreign bodies in the trachea and œsophagus.

In a typical examining booth, darkened, cases of sinus disease (antrum and frontal) were demonstrated by transillumination. In a second booth or room a cadaver was placed by means of which the diagnosis and management

of foreign bodies in the trachea and œsophagus was explained and illustrated. The recent instruments which have been invented for this purpose were shown and used.

Owing to the illness of Dr. Coolidge this demonstration was under the charge of Dr. Harris P. Mosher, assisted by Dr. H. A. Barnes.

9.—METHODS OF DIAGNOSIS BY MEANS OF THE CYSTOSCOPE IN GENITO-URINARY DISEASES.

In this demonstration five human bladders were used previously prepared, some of them in the usual Kaiserling solution, some in formalin-salt solution. Before hardening they were distended with the fluid, and bougies and glass rods tied in to ensure patent urethra and ureters. The difficulty in using these cadaver bladders is their inflexibility after keeping them in any solution that will preserve them. The bladders we used failed to show after even the Kaiserling treatment any serviceable preservation of color, but in other respects were satisfactory. They were slung in cloth-pockets in especially prepared boxes so as to give approximately normal relations. The cystoscopes were then used exactly as on a patient.

With the Nitze examining cystoscope the technique was demonstrated, the bladder wall shown, the ureters and a bougie tip protruding from the ureter opening into the bladder, etc.

With the Kollmann the special technique of its handling was shown with a demonstration of the location of the ureters and of foreign bodies placed in the bladder.

With the Nitze-Albarran catheterizing cystoscope we succeeded in showing to a good many spectators the actual technique of ureter catheterization. First the ureter opening was brought into the field of the cystoscope and shown to them in turn, then the catheter tip was protruded and brought in place to enter the ureter, this field showing the catheter tip directed to the ureter opening and ready to enter; both in the same field were then shown. Then the catheter was inserted and shown actually entered into the ureter.

With the air inflation direct view cystoscope we had found it impracticable to catheterize ureters on the specimens, but we demonstrated what can be seen through the

small brightly lighted field of this instrument, showing inserted foreign bodies, etc.

In addition to the distended specimens two opened bladders were shown demonstrating the ureter openings and their relation to the trigone.

The Harris segregator was shown in actual operation on a prepared bladder to the ureters of which siphon tubes brought a fluid supply from bottles on the table. This worked well and coupled with diagrams of the instrument seemed of interest.

The Newer Luy's separator was shown and explained. Unfortunately this instrument, being to some extent dependent on elasticity of bladder wall, cannot be made to work well in these prepared bladders.

The demonstration was successful in showing to a large number of the members of the Society some things unfamiliar to most of them and of real importance. Apparently to many the technique of bladder examination had been a very foggy matter and to these the demonstration of the feasibility of ureter catherization was something wholly new.

There was delay in getting started and some trouble later on from the difficulty in handling low-tension lamps with a street-current and somewhat unreliable controllers for such continuous use as this sort of demonstration calls for. This can be avoided next time.

The demonstration was in charge of Drs. F. C. Cotton and F. G. Balch, assisted by Drs. Binney, Chute and O'Neil.

10.—METHODS IN EXAMINATION OF URINE.

This demonstration was in charge of Dr. R. L. Emerson.

11.—METHODS IN BACTERIOLOGY.

The demonstration comprised the following:

Anthrax: A gelatine culture; Bacilli from a mouse dead of anthrax; Bacilli containing spores.

Tetanus: Photograph of culture. Bacilli with spores.

Actinomycosis: A culture of bovine actinomyces. Udder of cow with actinomyces (section). Smear of pus from liver of man showing actinomyces.

Glanders: Culture from horse, having passed through a guinea pig. Photographs of cultures. Bacilli from a culture.

Rabies: Section of Ammonshorn of rabid guinea pig and section of Ammonshorn of rabid dog (both showing "Negri bodies" the supposed cause of rabies). Numerous large photographs which were exhibited at St. Louis.

Diphtheria: Pseudo-Diphtheria: Xecosis: Pure cultures and throat cultures. Also the acid producing properties in serum-water-litmus, containing the sugars dextrin and saccharose. Microscopic specimens of same stained with methylene-blue and Neisser's stain. Preparations of toxin and antitoxin of diphtheria.

Bacillus of Dysentery: Shiga: Flexner: Duval: Cultures on agar agar. Also cultures in serum-water-litmus containing the sugars saccharose, maltose, mannit, dextrose, dextrine and lactose, showing the different acid-producing properties of the bacilli. Also microscopic specimens stained with dilute carbol-fuchsin.

Spirillum of Cholera. Finkler-Prior: Metchinkoff: Agar agar cultures. Also the indol production of the spirilla in peptone solution. Microscopic specimens stained with dilute carbol-fuchsin.

Stained Specimens: Pneumobacillus, showing capsule: Pneumococcus showing capsule: Streptococcus.

Cultures of: Staphylococcus pyogenes aureus; staphylococcus pyogenes albus; staphylococcus epidermis albus; staphylococcus pyogenes citreus; staphylococcus cereus flavus. Streptococcus pyogenes. Bacillus pyocyaneous; Pneumobacillus on agar; pneumobacillus in gelatine; Pneumococcus; Bacillus prodigiosus; Bacillus violaceus; Bacillus ruber plymuthicus; Bacillus rosaceus metalloides; Micrococcus roseum; Bacillus cinnabareus.

Tubercle Bacillus: Cultures of human, bovine, avian, fish and blind-worm races. Tubercle bacilli in sputum with method of straining. Tuberculin; its preparation and its use in the tuberculin reaction, with charts showing reactions. Homogeneous culture of Arloing and Courmont.

Diplococcus Intracellularis Meningitidis: Cultures on agar and blood-agar. Stained sediment of cerebrospinal fluid.

Typhoid and Colon Group: Cultures of typhoid, paratyphoid (2 races) and colon on agar, bouillon, glucose bouillon and litmus milk. Stained specimen of typhoid bacillus showing flagella.

Serum Reactions: Widal's reaction. Macroscopic agglutination test with preserved culture of typhoid bacillus. Tubercle bacillus agglutination test of Arloing and Courmont. Agglutination reaction of Shiga's bacillus of dysentery.

Blood Culture: Demonstration of technique.

Several cultures of gonococcus and smears of gonorrhœal pus stained by various methods were also shown.

Cultures of amœbae with typhoid bacilli grown on the Musgrave and Clegg media; stained specimen of the amœbae grown on the media; specimens of the amœba of dysentery in the unstained and motile state to show the appearance of the amœbae presented in the stools in amœbic dysentery. Methods of examination of stools for the diagnosis of amœbic dysentery.

The demonstration was arranged by Dr. H. C. Ernst. In his absence it was carried out by Dr. Langdon Frothingham assisted by Drs. G. L. Baker, E. E. Everett, H. W. Hill, F. W. Palfrey, H. J. Perry and E. N. Tobey.

12.—METHODS IN ESTIMATING COAGULATION TIME OF BLOOD.

Although several instruments have been used to determine the coagulation-time of the blood, only two are now in use,—(1) the Wright tubes, and (2) the Brodie-Russell moist chamber. Of these two, the Brodie-Russell is the more reliable.

In the demonstration before The Massachusetts Medical Society the Wright tubes were displayed, while the actual tests were made with two instruments modified respectively by Professor W. T. Porter and Dr. J. H. Pratt, from the original Brodie-Russell chamber. The tests consisted in the determination of the coagulation-time of fresh blood, drawn at the time from a volunteer, usually the visitor.

The determination of the coagulation-time of the blood is of value in the diagnosis, both of surgical and of medical diseases.

It is well known that certain patients suffering from jaundice die from fatal capillary hemorrhage after surgical operations. A knowledge of a delayed coagulation-time will allow the surgeon to decline to operate until an attempt has been made to restore the coagulation-time to normal. On the other hand, the medical value of the coagulation-time is seen in the differential diagnosis between Hemorrhagic Purpura and Hemophilia, since, in the former, the coagulation-time is normal, while, in the latter, the coagulation-time is markedly delayed.

The demonstration was in charge of Dr. A. H. Gould, assisted by Messrs. Jason and Charles Mixter.

13.—METHODS IN EXAMINATION OF BLOOD.

Methods of estimating hemoglobin and of counting the corpuscles, the method being illustrated by the actual preparation of specimens and by the use of charts. Differential counting, the preparation and staining of films and a demonstration of specimens stained to show the various corpuscles. Methods of counting the platelets and demonstrating these interesting, though as yet clinically unimportant bodies, in stained specimens were shown. A demonstration of the iodine reaction. The Deland hematocrit and its use in determining the volume index,—a method which bids fair to be of value in the diagnosis of pernicious anæmia, was also shown. Spectroscopic and chemical tests were shown. A show-case containing apparatus used in the examination of the blood was exhibited by the Sampson-Soch Co. Thanks are also due to Dr. R. C. Cabot and to Tufts Medical School for the loan of specimens.

The demonstration was under the general charge of Dr. R. C. Larrabee of Boston, assisted by Drs. R. E. Andrews, H. C. Dunn, H. C. Emerson, F. W. Gay and Mary W. Rowley.

14.—METHODS IN LEGAL MEDICINE.

This demonstration included illustrations of the physical character, chemical tests, microscopical tests and micrometry and the biological tests for blood.

A.—Physical character of stains.

1. Specimens illustrating the time of drying on polished surface (cardboard) and absorbing surface (pine board) of stains.

2. Specimens illustrating direction of blood spots and spatters, showing characteristic shapes (oval, oval with projection, indian club, and streak) of blood spatters made (*a*) by pulsating vessel parallel to surface on which spatters were found, (*b*) by pulsating vessel on surface held perpendicularly above, (*c*) by blood dropping from above, (*d*) by bludgeon striking a pool of blood.

3. Age of staining as shown by brownish discoloration.

4. Method of picking clots from cloth with dissecting microscope.

5. Method of making cells for microscopical study of corpuscles.

B.— Chemical tests.

1. Phosphotungstic acid test for collecting pigment from stains (especially washed stains). Not specific.

2. Guaiac test for blood—not specific.

3. Hemin crystals (specific).

C.—Microscopical tests and micrometry.

1. Diagrams illustrating at a magnification of 10,000 diameters:

(*a*) Types of corpuscles—mammalian (circular), non-nucleated, example (man); exceptions camel, llama (oval), non-nucleated, example (camel); fish, amphibian, reptilian, bird (oval), nucleated, example (chicken); lamprey (circular), nucleated.

(*b*) Relative sizes of mammalian red cells (averages from Gulliver and Wromley), including (in order of size) elephant, sloth, platypus, whale, camel, opossum, capybara, porcupine, monkey, kangaroo, wolf, woodchuck, rat, rabbit, bear, dog, hippopotamus, ass, hyena, mouse, mule, ocelot, bat, raccoon, squirrel (red), lion, tapir, squirrel (ground), ox, horse, pig, buffalo, cat, elk, leopard, sheep, goat, muskdeer.

(*c*) Variations in size of human red cells in health showing extremes in childhood (1–2385 in. to 1–7575 in. Hayem); extremes in adults (1–2900 to 1–2800 in. Hayem); ordinary variations (90% between 1–3100 and 1–3300 in. Formad); average (1–3250 in. Wormley), and average size of swollen spherical cells in aqueous fluids (1–4267 in. Formad).

2. Microscopical illustrations of:

(*a*) Types of red cells:

Mammalian—man; exception—camel; fish—goldfish; reptilian—snake; amphibian—toad; bird—chicken.

(*b*) Micrometry.

Filar micrometer (B and L) used with Leitz 1–16 oil immersion objective, giving a unit of 1–650,000 in. (actual limit of microscopic vision about 1–250,000 in.).

Scale micrometer (movable).

Stage micrometer (for standardizing eye-piece micrometers).

(*c*) Comparative sizes of mammalian red cells. (Microscopes supplied with disc micrometers for comparison).

Smears of guinea pig, muskrat, monkey, rabbit, gray squirrel, dog, mouse, ox, horse, pig, cat, sheep and goat bloods.

Specimens of normal human blood, shrunken spherical cells in aqueous diluent, menstrual blood showing squamous cells, and blood from stain. Smear of blood from chlorosis showing variation in size of cells from 1–2389 in. to 1–5385 in.

D.—Biological tests.

1. Diagrams illustrating hemolysis (as characteristic of biological reaction).

2. Method of preserving anti-body producing substances, such as blood, in a sterile condition. (Loeffler.)

(*a*) Human blood dried on a glass plate.

(*b*) Human blood sterilized and sealed in a glass tube.

3. Method of humanizing rabbits.

(*a*) Collection of human placental blood in sterile flask.

(*b*) Cards illustrating injection records and weights of rabbits.

(*c*) Blood obtained by cardiac puncture from rabbits.

4. Precipitation test.

(*a*) Description of technique, character of reaction, and sources of error.

(*b*) Series of animal bloods on filter paper for controls.

(*c*) Individual, graduated capillary, pipettes for diluting each blood tested.

(*d*) Cubic centimeter tubes and racks (Nuttall).
(*e*) Qualitative tests with human blood (early-clouding).
(*f*) Qualitative tests—precipitate, collected in capillary tubes for measurement and comparison.

5. Agglutination test—as obtained by adding to solution of stain, heterologous blood (not specific, only valuable as confirmatory of precipitation test).
(*a*) Specimen showing agglutination of human blood by rabbit's blood.

6. Hemolysis—not specific enough for medico-legal purposes.
(*a*) Smear of human blood on plate of glass with letters M.M.S. formed by solution of red cells by hemolytic serum of a humanized rabbit.
(*b*) Microscopic illustration of solution of red cells (human) by hemolytic serum (humanized rabbit).

The demonstration was given by Dr. Timothy Leary.

15.—DEMONSTRATION OF THE METHODS OF KEEPING MEDICAL RECORDS.

The demonstration consisted of the display of either specimen records or record blanks to illustrate what seemed to us the most approved methods of keeping the following:

1. Private Records.
   (*a*) Cases.
   (*b*) Accounts.
2. Hospital Records.
   (*a*) Clinical.
   (*b*) Pathological.
3. Medical Literature.

A careful study of the subject has convinced us that in all cases of the above groups the most convenient and accessible method is by the use of index file, and consequently the entire exhibit was devoted to it in its various forms. In general, the advantage of this method over all others lies in its unit character, which admits of (1) convenient arrangement according to any desired plan, (2) unlimited growth, (3) great accessibility, and (4) any desired scheme of indexing.

Several types of private records were shown, including cards of various sizes,—large for office work, and smaller

ones of such size to be conveniently carried in the pocket or bag. In every instance it was proposed that they should ultimately be arranged in an index file of suitable form. Similarly, a large variety of cards ruled for accounts (mainly 4 x 6 ins.) were included in the exhibit. A considerable display was also made of hospital records, consisting, for the most part, of large sheets printed in forms for special purposes, according to the type of hospital. In some cases these were ultimately bound into volumes, in others filed in appropriate cases, always with separate index files, by name, disease, complications, etc. During the patient's stay in the hospital, all records, charts, photographs, etc., should be kept in a folder either at the bedside or in some accessible place.

The system when applied to the keeping of medical literature admits of the filing of abstracts of articles on card catalogue cards, or of the articles themselves by title with a cross reference catalogue by authors and subjects.

16.—METHODS IN EXAMINATION OF SPUTUM.

The sputum demonstration consisted of microscopic preparations, cultures and photographs of some of the more important findings in diseases of the respiratory tract.

Besides tubercle bacilli and actinomyces, sputum preparations were shown, illustrating infection with influenza bacilli, micrococcus catarrhalis, pneumococci, pseudo-pneumococci, etc. It is important to recognize that these organisms are capable of causing mild or severe respiratory infection, the clinical course of which in some instances may resemble pulmonary tuberculosis.

Elastic fibres, at times valuable in the differentiation of pulmonary abscess from bronchiectasis, and conclusive evidence of pulmonary destruction, whether of tubercular or other origin, were demonstrated. Fibrinous casts of the bronchi and Curschman spirals were also shown.

The danger of the spread of tubercular material through flies was illustrated by sections of flies and microscopic preparations of their excrement, showing tubercle bacilli. That infected fly specks may continue to be a danger after their deposit was shown by the preserved organs of a guinea pig, in whom tuberculosis had resulted from the injection of such material after it had dried for fifteen days.

Several specimens of Distoma Ringeri, the cause of an hæmoptysis, endemic in Japan, Corea and certain parts of China, were a feature of the exhibit. They were kindly sent for the demonstration by Prof. Inouye of Chiba, Japan. The cases may resemble tuberculosis clinically, but the diagnosis can be made by finding their eggs—preparations of which were shown.

The demonstration was in charge of Dr. F. T. Lord, assisted by Dr. Wm. H. Smith.

17.—METHODS OF STUDY OF BLOOD PRESSURE.

In the blood-pressure demonstration the instruments of Riva Rocci, Stanton and Janeway for estimating the blood-pressure were shown and their principles explained. Visitors were given an opportunity to familiarize themselves with the technic of these instruments by taking a patient's blood-pressure.

The inflatable rubber suit devised by Dr. Crile to counteract the loss of the vaso-motor tone in cases of shock was also exhibited and demonstrations given of the efficiency of the procedure by raising and lowering the blood-pressure of a patient.

Blood-pressure charts taken on cases in the Massachusetts General Hospital were on exhibition to illustrate the characteristic curves and levels of nephritis, surgical shock, cerebral compression, etc.

This demonstration was in charge of Dr. F. T. Murphy, assisted by Drs. J. B. Ayer and S. B. Vincent.

18.—METHODS IN CYTO-DIAGNOSIS.

There were demonstrated specimens of pleural and abdominal fluid, stained preparations of the sediment of pleural and cerebro-spinal fluids, including acute infectious and tuberculous varieties, and also transudates were shown as well as the apparatus, reagents and stains necessary for making the preparations.

A guinea pig showing tuberculous lesions of the liver, spleen and lymph nodes as a result of previous inoculation with tuberculous pleural fluid was exhibited.

A collection of photographs illustrating the different characteristics of the various sediments and different cells occurring in the different varieties of pleural effusions was shown.

The purpose of this exhibit was to demonstrate that the examination of serous fluids was of aid in determining their etiology.

The demonstration was in charge of Dr. Percy Musgrave, assisted by Mr. Roger Lee.

19.—METHODS IN GASTRIC DIAGNOSIS.

Patients suffering from different gastric diseases were shown, physical examinations made, and where it was practicable chemical and microscopical examinations were made of the stomach contents, demonstrating the modern methods employed in this work.

A large number of patients were present, showing the different diseases of the stomach from simple catarrh, an acidity, hyperacidity, retention of food, gastroptosis, etc., to malignant disease.

Some patients with œsophageal diseases, viz. stricture, diverticulum, were also shown.

The demonstration included some cases of malignant disease indicating operation, and other cases which had been previously operated upon for like conditions.

The demonstrations were given by Drs. Franz Pfaff and Dr. Elliott P. Joslin, assisted by Drs. M. Vejux Tyrode, Richard F. Chase, Henry T. Barstow, Wm. L. Smith, Louis Kekm and Malcolm Seymour.

20.—METHODS IN EXAMINATION OF FECES.

The demonstration of the clinical methods of examining feces was divided into five sections.

1. Microscopical appearance of the stools in different pathological conditions and their diagnostic significance. A collection of specimens for this purpose had been especially prepared. Portions of stools had been placed in large test-tubes, a small amount of formalin added and the tube sealed. The appearance and color of the fresh stool was preserved. In this collection were stools dark from admixed blood, and stools equally dark that were free from blood. The similarity in appearance emphasized the necessity of performing the chemical test for blood. Pea-soup discharges of typhoid fever, green stools containing bilirubin and biliverdin, and stools composed largely of fat crystals, and one consisting almost entirely of fat in the form of oil droplets, were shown among others.

II. Bacteriological diagnosis of typhoid and dysentery from a study of the stools. The method of isolating the typhoid, differentiating colon, and dysentery bacilli, by the use of Hirs's media, was shown. By this method the bacteriological diagnosis of typhoid infection can be made in fourteen hours. The agglutination tests with the dysentery bacilli were performed.

Collection of interstitial parasites, and the methods of identifying them. Diagrams of parasites were prepared to show clearly the chief diagnostic features.

III. Special attention was drawn to the dibothriocephalus latus. Imported cases are more common than is generally supposed. Eggs and segments of worms were shown under the microscopes.

IV. Chemical methods of diagnosis. The guaiac test for blood was demonstrated continuously throughout the morning. It was thought advisable to show clearly and repeatedly a simple modern test that would be of value to the general practitioner, and the guaiac and aloin tests for blood were selected. Three demonstrators were engaged in performing these tests.

V. A collection of the standard works on the clinical study of the fæces was shown.

The demonstration was in charge of Dr. J. H. Pratt, assisted by Drs. A. C. Cummings, J. W. Dewis, C. W. Duval, W. R. P. Emerson, H. C. Low and H. W. Thayer.

21.—METHODS IN NEUROLOGY.

A number of patients were shown with different disorders of sensibility, motion and muscular nutrition and the various methods of diagnosis demonstrated on them. The different groups of visitors were passed along from one demonstrator to another, and thus given an opportunity to see the methods of examination. On a model from the Massachusetts Normal Art School, the different areas of the skin corresponding to the peripheral-nerve distribution and the nerve-root distributions respectively were mapped out and demonstrated.

The demonstration was in charge of Dr. J. J. Putnam, assisted by Drs. Knapp, Prince, Taylor, Walton and Waterman.

22.—METHODS USED IN BOARD OF HEALTH EXAMINATIONS.

I. Demonstration of the Widal or agglutinative test for typhoid fever. This consisted in showing the methods used in the laboratory for testing blood dried on bits of weighed paper and sent by mail from different parts of the State. The paper is weighed before it is sent out and again when received with the dried drops of blood on it. The difference in weight equals the weight of dried blood. This is cut out and soaked in a given quantity of a bouillon culture of typhoid bacilli so calculated as to yield a dilution of one of liquid blood to twenty of culture fluid, or about one of blood-serum to thirty-five or forty of culture fluid. Clumping after two hours is considered a positive test for typhoid.

The specific action of typhoid agglutinin was demonstrated as follows:—Two rabbits were immunized by repeated injections of heated cultures of typhoid and paracolon bacilli respectively. The blood serum of these rabbits diluted from one-twentieth to one-two-thousandth was distributed in small test tubes and the bacilli added from cultures. Marked agglutination of typhoid and paracolon bacilli in their respective sera was evident up to one-one-thousandth, whereas there was no recognizable clumping when paracolon bacilli were added to "typhoid" serum and typhoid bacilli to "paracolon" serum even in dilutions as strong as one-twentieth.

II. Demonstration of the method used in making a diagnosis of malaria. Films of blood dried on coverslips and sent in special tin capsules by physicians from different parts of the State are fixed and stained and carefully examined for malaria parasites.

III. Demonstration of leprosy bacilli obtained by swabbing the nose of two cases of this disease in Massachusetts. The smears are made on slides or coverglasses in the usual way, and stained according to the methods in use for tubercle bacilli.

IV. Demonstration of the method used in making a diagnosis of diphtheria. The following were shown:

Outfit for taking cultures in cases of suspected diphtheria.

Cultures from throats suspected of containing the bacilli of diphtheria.

Pure cultures of bacillus diphtheriæ, staphylococcus pyogenes aureus, streptococcus pyogenes.

V. Demonstration of the method used in the diagnosis of tubercle bacilli in sputum.

Outfits were shown in which the sputum is collected by the patients and sent by physicians from various parts of the State to the laboratory at the State House. The method of staining and examining dried films of sputum was shown and pure cultures of tubercle bacilli exhibited.

VI. The staining and microscopic examination of cerebro-spinal fluid, obtained by lumbar puncture, were also shown.

The Boston Board of Health Laboratory Exhibit was designed chiefly to demonstrate the modifications of ordinary bacterial methods of diagnosis devised and used to meet requirements of public health practice in which large numbers of examinations from different sources must be rapidly made without sacrifice of accuracy or efficiency. These included special forms of apparatus, large slides, special water-baths, staining baths, decolorizing baths, needle holders, etc., all co-ordinated to this end. The outfit devised for the collection of the specimens for examination from cases suspected of diphtheria, tuberculosis, typhoid, malaria and glanders, were exhibited. Certain new devices were shown, such as a water-pressure filter for securing bacteria-free filtrate from liquid cultures; porous tops for Petri dishes, used to prevent spreaders in milk and water plate counts; an apparatus for staining bacteria or other minute bodies while under continuous direct microscopic observations, etc.

These demonstrations were under the direction of Drs. Theobald Smith and H. W. Hill, assisted by Dr. A. L. Reagh, Mr. B. R. Rickards, and Mr. E. L. Walker.

23.—METHODS IN COMPARATIVE PATHOLOGY.

I. Demonstration of the resistance of red corpuscles of the horse to different strengths of salt solution and diluted serum. The demonstration is carried out by putting salt solution in different strengths from 0.40 per cent. to 0.68 per cent. into small test tubes and then adding sedimented red corpuscles. The amount of hemolysis or laking is

measured by a freshly prepared series of tubes containing hemaglobin tints of definite per cent. At the same time, to tubes of serum definite amounts of distilled water are added, and red corpuscles are then added to these to determine the amount of water which can be added before laking occurs. The demonstration shows:

1. That in blood the red corpuscles can be divided into a large number of classes of different resistance to salt solutions and to diluted serum.

2. That each horse has red corpuscles of definite degrees of resistance which are constant for the same individual but which may differ from those of another horse, and that resistance varies within certain limits.

3. That horses which have been bled many times may or may not show a diminished resistance of the red cells.

4. That from a comparison of the hemolysis in salt solution and diluted serum may be calculated the osmotic tension of the serum.

II. Demonstration of a series of cultures of tubercle bacilli from man, horse, cattle, pig, dog, cat, *Nasua narica*, and pigeon.

The demonstration was in charge of Dr. Theobald Smith, assisted by Mr. H. R. Brown.

24.—METHODS OF RAPID ANATOMICAL DIAGNOSIS.

This demonstration consisted in an exhibition of a rapid method of preparing sections of tissue for microscopical examination by means of freezing with carbon dioxide gas.

The method is especially useful in the diagnosis of tumors and other pathological tissue, because by it stained and permanently mounted sections satisfying all the usual requirements may be obtained in a few minutes.

The demonstration was in charge of Dr. James H. Wright.

ARTICLE XIV.

THE ANNUAL DISCOURSE.

THE GENERAL PRACTITIONER AND THE SPECIALIST.

By JOHN L. HILDRETH, M.D.
OF CAMBRIDGE.

DELIVERED JUNE 13, 1906.

NOTE.—At an Adjourned Meeting of The Massachusetts Medical Society, held Oct. 3, 1860, it was

Resolved, "That The Massachusetts Medical Society hereby declares that it does not consider itself as having endorsed or censured the opinions in former published Annual Discourses, nor will it hold itself responsible for any opinions or sentiments advanced in any future similar discourses."

Resolved, "That the Committee On Publications be directed to print a statement to that effect at the commencement of each Annual Discourse which may hereafter be published."

THE GENERAL PRACTITIONER AND THE SPECIALIST.

Mr. President and Fellows
of The Massachusetts Medical Society:—

The modern tendency toward subdivision and specialization is very strong. It is manifest in education, where the elective system which permits the selection of particular studies to suit a special training or individual caprice extends far back into the preparatory schools and reaches forward through the whole period of preparation for the active work of life. It appears in industry. The watchmaker no longer makes a complete watch or the shoemaker a complete shoe. Each is engaged upon some swift and deft machine which turns out parts of the complete product to be fitted to other parts by other hands. The student of science specializes. He no longer attempts a comprehensive understanding of the whole realm of knowledge, but early picks out his small chosen bit of the field and devotes his life to an assiduous cultivation of it. The successful merchant no longer tries to direct all the details of his business. These are subdivided and entrusted, this to that confidential representative, that to this head of a department. So through all domains of activity.

This tendency is nowhere more manifest than in the medical profession. So great are the changes which it has brought about that they are imperfectly realized even by the members of the profession. In general preparation for

the profession there has been a remarkable advance during the last forty years. The difference in the amount of work required to graduate from the better medical schools now and forty years ago is very great indeed; and the character of the men entering the profession seems to me much stronger and better fitted for the scientific and successful practice of medicine now than then. Then bacteriology was unknown; medical chemistry and human physiology were inadequately taught; experience counted for everything with a doctor, and scientific attainments for very little. With this change in preparation for the profession there has come a change equally marked in the practice of it. Formerly, as I have said, it was experience that counted as the prime requirement in a doctor, and that was gained in large hospitals in the medical centres of our own and other countries.

There were then no specialists except those who devoted themselves to the eye and to the ear. There was, it is true, a recognized distinction between physicians and surgeons, but the innumerable classes of specialists that exist to-day were unknown and undreamed of.

A part of the present subdivision of work has come about doubtless, through the adoption of German methods of study and instruction in medicine. The German mind is content to expend its energies upon detailed work, to pursue a subject to the farthest possible boundary, zealous rather in the search for ultimate causes than in the practical application of knowledge. But whether from the extension of the German influence or from other causes there has come about the existing state of things, with specialists for every organ of the body. Moreover, with this extension of specialism, there has come to pass a marked change in the position and possibilities of the general practitioner. The relations of the two—the general practitioner and the specialist—to each other and to the community furnish an

interesting and, I hope, not unprofitable subject of study, to a hasty and inadequate discussion of which I venture to ask your consideration.

And in the first place, let us look for a moment at the advantages which each class of practitioners enjoys, and the disadvantages under which he labors. Neither path is wholly strewn with roses. Success may be won in either, and the rewards of success are great; but they come only as the fruit of labor and sacrifice.

The work of the general practitioner is harder to-day than it was a generation ago. He is much less independent, and he has less time to himself. The multiplication of telephones makes him more subject to interruptions by his patients, who find it so easy to call him by day or by night, to ask questions—some necessary, some unnecessary—that they leave him no time which he can call his own. He is on call at all times, and it is not surprising that, while he appreciates the uses of the telephone, he sometimes regards it as a device of the Evil One. He is a slave to the peremptory call of the instrument, and must constantly be ready to answer it, as in many cases no one can answer it for him. The telephone, and that later invention, the automobile, each of which, in its way, annihilates space and time, greatly affect the general practitioner in another way. They increase competition by making available for a patient, who for any reason has become im-patient, the services of a physician from a distance. These new appliances, and the constantly enlarging necessities of office outfit and supplies, add greatly to the expenses of the general practitioner. Altogether, it would be fair to estimate the increase in the expenses of the regular practitioner during the last thirty-five years at least 25 per cent. If he is to be, as his patients say, "up to date", he must attend medical meetings, read the medical journals and treatises, visit the hospitals or have a hospital service of his own, and from

time to time must see what is being done in his profession in other countries.

All these things make the general practitioner's life much more laborious than it was a generation ago. The simple life of the community has also disappeared, and a very strenuous and complex life has taken its place. The practitioner has to adapt himself to these conditions. He must be alert and constantly on the watch to see that no one does better work than he, and whilst he does not advertise or solicit patients, he must make himself desirable enough to ensure his being sought for. Very few general practitioners who have been away from schools and hospitals for fifteen years and are thoroughly established have a moment's time to themselves, from the time that their vacation ends in the summer or fall until it begins the next summer. From early morning to late at night they are in the harness; some one wants them, wants them in a hurry. If they want a half day off for leisure, some very sick patient prevents. If they plan for a day's recreation in the country, it is more than likely that some patient or expected patient will detain them. All these conditions are much changed from those of thirty or forty years ago.

Another important change affects the lot of the general practitioner. The medical profession has come to feel that everything that is brilliant, everything that makes an impression upon the laity, must be something that can be seen or that can be touched with the hands; in other words, must be something of a surgical nature. The man who gives advice concerning a difficult problem in medicine, who helps to complete a diagnosis when the symptoms are complicated and an accurate diagnosis extremely difficult, does very little to impress the laity by comparison with the man who performs a surgical operation. The conditions are wholly reversed from those of the time of the "barber surgeons" when the surgeon sat at the feet of the family phy-

sician. Now the surgeon is to be pacified only by the homage and admiration of the general practitioner.

The advantages and disadvantages of the specialist are obvious. He has regular hours for his office and regular hours for the hospital, and his day's work is done with the day. He can study to the farthest bounds of knowledge the causes and treatment of the diseases to which he has devoted himself. The field is not too large and the problems, although often intricate and sometimes unsolvable, are not too many. But he has to give more years to get a profitable following and he is less sure of success in the end. His time of life work is shorter. He becomes old-fashioned and some young man fresh from the large hospital fields of Europe becomes the public favorite in his stead. The field for the successful specialist is restricted to the large centres of population. A few are found in cities of from 50,000 to 100,000 inhabitants, but their opportunities for a large practice and remunerative fees are small. One thing which the specialist misses cannot be estimated by any pecuniary standard. He gains warm friends and admirers among his patients, but he does not know them as the family physician knows them; he is not their god-father, nor their elder counsellor and trusted friend as the family physician is. There is no experience in life which is quite like the peculiar and satisfying relationship which exists between the family physician and the family patients. The weary doctor derives a pleasure and comfort from this which no fee or words of praise can approach, in value.

Under existing conditions, the general practitioner cannot be expected to do original work. Everything of that nature,—everything that promotes the progress of medicine in a scientific way, must be done by the specialist. But the general practitioner must be alert to follow all new discoveries and processes and to avail himself of them. It is a natural consequence of the changed conditions that young

men entering the profession are drawn toward specialism rather than the general practice of medicine. My observation is that general practitioners who have sons in the profession encourage them, not to follow in their own footsteps, but to become specialists. I do not recall a single instance where the reverse is true, and where a father who is a specialist has a son who is a general practitioner. One of the older physicians of my acquaintance once said to me that it took a man of iron constitutution to follow general practice, and he had on that account led his son to adopt a special form of surgery.

One strong reason which impels men to become specialists is not avoidance of night work and the other hardships of the general practitioner, but the increasing demand of the people for specialists. More and more it is coming to pass that any one who has or fears that he has some organic trouble asks his family physician "Who is the best specialist in this or that?" or goes to a specialist without asking. It is in response to this tendency that we find that those doctors who advertise extensively, and who, while they may legally be practitioners, are not recognized by the profession, invariably advertise as specialists, with infallible cures for some particular malady. The story is told of a young lady who was asked to do something, and who, taking out her engagement book, said plaintively that she would have to be excused for that week, as on Monday she was to visit her oculist, on Tuesday her aurist, on Wednesday the orthopedic specialist regarding the arch of her foot, on Thursday she must go to a specialist in pelvic disorders, on Friday to a specialist in nervous disorders, and on Saturday to a specialist on the spleen.

It has always seemed to me that, when a general practitioner thinks that a patient should have the advice of a specialist, he should again, as he no doubt has done before, give his patient as thorough an examination as if he had

never seen him, so that nothing may be overlooked and he may have as complete a knowledge of the case as possible, and be sure that his decision is correct. In this way some new light may be let in upon the diagnosis, and some help secured for the treatment before the decisive step is taken of recommending the patient to go to a specialist.

It has not often fallen to my lot to be present when a specialist first saw his patient. It has not been my invariable custom, I regret to say, to send a detailed statement of the history of a case and its treatment to the specialist, so that he might have the advantage of all my knowledge of the case at the time that the patient reaches him. I suppose that it does not matter what the case is,—whether of the eye, or ear, or brain, or abdomen, or skin, or whatever else,—it must be that all thorough specialists make all inquiries for themselves at the first visit, just as though the patient had not been seen by any other person and just as though they had no information regarding the case from the practitioner sending the case to them.

But I am quite sure that some of the specialists are not thorough in their examination of cases, but simply accept the patient's statements, and examine him for the trouble for which he has been sent to them. I have seen some very sorrowful mistakes made in this way by specialists. On the other hand, I have seen some remarkably good work done by specialists who have given the patient a thorough looking-over, have gotten a complete history of the sickness, and have taken sufficient time to master all the details of the case up to the time when it presented itself for their treatment.

Some specialists have the excellent custom of writing out their opinion of what the diagnosis is, and what they believe to be the proper method of treatment, and of sending this statement, very soon after they have seen the patient, to the practitioner who sent him to them. This is helpful

to the general practitioner; it brings the specialist nearer to him; it makes the treatment of the case more satisfactory if the work of the specialist seems to go along as a continuation of that of the general practitioner. I have been greatly helped by such letters, and I believe that if this were a more general practice it would be of advantage to patient, general practitioner and specialist alike.

And I believe also that it would be helpful if the specialist, when he is through with a patient, should send him back with a note to the general practitioner, so that he might see what the result of the treatment was, the specialist practically turning the patient back to the general practitioner, so that conditions should be restored which existed before the family physician sent the patient to the specialist. Perhaps this is done oftener than I suspect, but I am sure that a general observance of this custom would break down the barrier which now often exists between the general practitioner and the specialist. The general practitioner would not be afraid of losing his patient for good if this practice were followed, and he would be the gainer by knowledge which might be of help to him if a similar case should present itself. These may seem trivial points of etiquette and of very little consequence to a busy man; but if the work of the profession is to be thoroughly and carefully done, such co-operation between the general practitioner and the specialist is very desirable, especially in difficult and baffling cases.

There has come, I am sure sometimes, to be some sense of rivalry between the general practitioner and the specialist. The general practitioner is sometimes inclined to feel that, if he sends his patient to a specialist, the latter will treat lightly what he has done for the patient, and will alienate his regard. Also, he may feel that the specialist is prompted by pecuniary considerations and thinks chiefly of his fee. Some general practitioners, for these and simi-

lar reasons, hesitate to send patients to specialists perhaps delaying to the detriment of the patient. This is all wrong. For professional, humanitarian and scientific reasons, there should be accord between the two. Each should recognize and respect the other as doing good work in his line and should co-operate with him in every way possible. These rivalries and misunderstandings are more likely to develop in the smaller cities and towns than in larger medical centres; but wherever they exist, and to whatever causes they may be due, all who have the highest interests of the profession at heart should allay and relieve them. If the general practitioner is annoyed by the greater prosperity of a specialist, he should recollect that the increasing demand for specialists is in line with the tendency toward specialization in the other professions and in business; and also that in a considerable number of cases, the people who seek specialists do not have family physicians, but are led by what they hear about a specialist or read about him in the newspapers to seek him when they have or think they have an ailment which comes within his special field. The specialist nowadays does not receive his patients from the family physician in anything like the proportion in which he formerly did.

The general practitioner, who spends all his time in looking after $3.00 ailments, thinks of the specialist as sitting in his office gathering in $5.00 or $10.00 fees; or as engaged in surgical operations for which he receives large compensation. But he forgets that, while the specialist may spend his afternoon hours in this profitable and pleasant fashion, he has devoted his morning to hospital work without remuneration. Though, to be sure, his afternoon may bring him larger returns than the entire day of the general practitioner brings to him. From this view-point of the general practitioner, the work of the specialist may well seem to be more on a commercial basis than his own,

not only because of his larger fees, but because often he sees his patient but once, and there is an entire absence of the personal tie which is so strong between the family physician and his patients.

As commercialism has taken so strong a hold upon other callings and is sometimes not altogether absent from the movements of the clergy, it would not be strange if its influence were felt among specialists. They are in a way of knowing as much as any one of the rapid accumulation of large fortunes in other vocations. The papers are full of facts regarding these fortunes and the processes by which they are heaped up. Doctors are quite pardonable if they are not exempt from some aspirations in this direction. The change in habits and expense of living which has taken place among the people with whom the doctor associates makes it extremely difficult for him to maintain his proper social level. The cost of educating his children and establishing them in life has increased greatly; and, both by reason of these heavier burdens and of the lower rates of interest on investments, the money which it is necessary for him to lay by to provide for them and for his own old age is much larger than was required a generation ago.

There is, I am sure, a feeling among some of the older men in the profession that the spirit of commercialism is taking a very strong hold of the whole profession; and that, even in conservative Boston, where the highest professional standards have been maintained, there is danger that the central idea of relieving human suffering and making the lives of our fellow beings more pleasurable and more peaceful may give place, to some extent, to consideration of the size of the fee which the doctor may exact from his patient. In too many instances this fee seems to be based,— as the railway magnate said of the freight charges, upon "what the traffic will bear."

When the Prince of Battenberg visited this country some

months ago in command of a British squadron, and as the guest of our government, he carried away, as a souvenir of his visit, the receipted bill of a New York dentist for $1,000. as the price of filling four teeth. The distinguished guest was not in a position to remonstrate; he paid the bill like a gentleman, and it was only by an accident that its extortionate character was made public. But, when the facts were made known, they aroused just indignation in the public mind, and more than one rich and patriotic American would rather have paid the bill from his own pocket, had that been possible, than to have had the distinguished guest carry away such a reminder of American rapacity.

But the charge of this New York dentist is paralleled by many charges for surgical work. Recent correspondence in one of our medical journals contains an instance of this sort.

A New York surgeon asked $1,000. for an operation for removing an appendix. The mother of the patient offered $600. but the surgeon protested, cited testimony of brother physicians to prove that the fee was not excessive, and finally received the balance. The letter which enclosed the last check was as follows:—

"My discussion with you has been a friendly one, and so you will not, I am sure, suspect me of any acrimony, when I say that my feeling about the present excessive charge of surgeons is a very general one, and the reflection of a sentiment that is everywhere one of surprise and dissatisfaction. We do not question your ability, but we feel you make us pay too dear for it."

This fee was not exceptional. Higher fees have been charged for similar services; and one case is on record where a surgeon charged his patient,—a very rich man,—$15,000. for an appendix operation, the case being one of no special difficulty, and the charge, evidently, being

assessed with reference to the patient's wealth rather than the service rendered him. Possibly the surgeon may have been a bit of a Socialist in his way, and may have taken this method to assist in the redistribution of wealth. I am quite sure from considerable personal observation in the Mississippi Valley and in the far West, and on the other side of the Rocky Mountains, that this spirit of commercialism has gotten a pretty strong hold upon the profession. Medical and surgical service is considered and estimated according to its money value to the giver of the service. So far has this gone in some of the western cities that the fee tablet has in some instances of which I have a personal knowledge contained nearly two hundred items where the price was fixed for certain services, and where the whole method in which the price was varied and fixed to the service rendered seemed to be based on the principle of exacting from the patient as much as he could possibly pay.

There are considerations on the other side of this question of fees to which it is too much to expect the laity to give much heed. As has been truly remarked in the discussion of this question, all eminent surgeons have undergone a rigid and severe technical training after their graduation, and it is this training which makes their hand sure while operating. The patient pays them, not for the brief time spent in an operation, but for the skill and training which have been acquired by years of strenuous effort, and upon which he bases the assurance with which he gives himself to the surgeon's knife, certain that everything which science can do to relieve his suffering and save his life will be done. That sense of assurance is worth so much to the patient that he should not begrudge paying for it, provided the fee of the operating surgeon is not beyond reason. There is another consideration also which the laity overlook. Every surgeon and every physician devotes much time to hospital cases or to cases among the poor, where only

nominal fees can be asked at the most, and where often it is impossible that even these should be paid. In no other profession is a man expected cheerfully to take such risks and undergo such hardships at the dictates of humanity, without expectation of reward. If a physician or surgeon charges a wealthy patient a large fee, it may be in order that he may thereby be the better able to render gratuitous service in the name of humanity.

No one will deny that the doctor who gives many years to study, who deprives himself of many of the pleasures of life that he may minister to suffering humanity, should be properly remunerated so that he may have the ordinary comforts of life. He should be able to make all the usual provisions against sickness and distress, whether to himself or his family, to educate his children and to enjoy an old age free from worry and care. But he is not like the man engaged in business who studies every transaction to know just how much profit it is to yield him financially. He should study every case, not from the standpoint of dollars and cents, with a view to making the utmost possible by multiplying his visits or playing upon the fears of his patient, but with a view to doing everything in his power to relieve and save his patient without the slightest regard for the compensation he may receive.

Before dismissing this subject of commercialism in medicine, I want to add a word concerning a certain class of practitioners, constituting a considerable percentage of the brotherhood, who are at the farthest possible remove from any reproach on this score.

Most of those whom I have in mind live in the country towns, where business methods do not prevail as in the larger centres of population. In these communities, long credits are almost invariably given, accounts between neighbors and friends sometimes run indefinitely without being brought to a settlement, and bills for groceries, provisions,

dry goods and other articles for family use are not paid every month, or every three months, and sometimes not every year. Naturally enough, the doctors fall sometimes into the same habits that the farmers and mechanics and laboring men among whom they live have acquired. Assuredly these men do not need a warning against carrying on the practice of medicine for the purpose of accumulating a fortune. Their life is hard, their hours of labor long. The ready money in their communities is limited, and they have to adapt themselves to the conditions around them. They suffer more than any other class from these conditions, because in these rural communities the doctor's bill often is the last to be paid. If they have children growing up and to be educated, they find it extremely hard to do for them what they would like to do.

I believe that, as a class, these men are the most self-sacrificing, honest, conscientious workers for the relief of human suffering in the whole range of the medical profession. But I wish that they might adopt new methods, especially in the management of financial matters. It would be better for them, and better for the profession, and better for their patients, if they were to send their bills promptly once a month or once in three months. Their fault is a fault which leans to virtue's side, and it is a great deal more lovable than the fault of commercialism, which lies at the opposite extreme. But I wish that these faithful servants of the public might do better for themselves. If they would do even half as well for themselves and their families as they do for those to whom they minister, their outlook would be much brighter than it now sometimes is when some accident or sudden sickness curtails their activities. And, although they do not realize it, a change in their methods would be of advantage to the profession by dignifying its proper compensations and preventing the spread of the destructive notion that a doctor's bill is something

that can be attended to at any time,—or not at all, if it is not convenient.

"Noblesse oblige" is a familiar French phrase. When the French say this, they mean that nobility of birth imposes a special obligation upon whoever boasts of it. The man nobly born cannot be, or at least ought not to be, like common men. The very circumstance which increases his privilege increases in the same ratio his responsibilities. But, if this is true of noble birth, how much more is it true of a noble profession.

It is the work of the physician to relieve suffering; to calm apprehension; to make easy and secure the entrance into life; and to attend humanity every step of the way from the cradle to the grave. His it is to study and aid the processes of Nature in restoring health; to provide a just ground of confidence in time of accident or sudden fear; to be a friend, counsellor and helper in every physical emergency of life for which human skill is of any avail; to do all this gladly, enthusiastically, unselfishly. The annals of the profession are full of the records of men who have faced every contagion and every form of peril without a thought for their own personal safety. In every great epidemic many doctors have given up their lives who might have saved themselves by flight but who would not leave those who needed them. On every battlefield, in the track of advancing armies, surgeons have cared for the wounded while bullets were still flying. This very year, doctors have been murdered by the side of their patients in Russia, because they would not forgo their work of mercy at the demand either of Cossacks or of the mob.

This is a noble profession to which we belong. Richer than any heritage of birth is that which falls to us as successors of the courageous and self-forgetful practitioners of the past. The nobility of this heritage imposes upon us a corresponding obligation. We cannot be, we ought not

to be like men without such a heritage. If the mercenary were to be substituted for the humane motive in the practice of medicine, the most ennobling and beautiful qualities of the profession would be lost.

It is a serious misfortune that such a profession as ours should even become subject to the suspicion of commercialism. But, if it should yield to that influence, if it should be overwhelmed by it,—that would be a calamity indeed.

ARTICLE XV.

THE SHATTUCK LECTURE.

A CONTRIBUTION TO THE CHEMISTRY OF THE BACTERIAL CELL AND A STUDY OF THE EFFECTS OF SOME OF THE SPLIT PRODUCTS ON ANIMALS.

By VICTOR C. VAUGHAN, M.D.
OF ANN ARBOR, MICH.

DELIVERED JUNE 12, 1906.

A CONTRIBUTION TO THE CHEMISTRY OF THE BACTERIAL CELL AND A STUDY OF THE EFFECTS OF SOME OF THE SPLIT PRODUCTS ON ANIMALS.

PART I.—GENERAL STATEMENT.

Introduction.—For some years past the research work carried on in my laboratory has been along the line of the chemistry of the cell. The large tanks which I devised for growing bacteria in mass have enabled me to obtain cellular substance in large amount and free from extraneous matter. The results reached in the study of the material thus obtained have been given in contributions to current medical and scientific literature published from time to time by my students and myself. Inasmuch as this investigation has been quite different in conception and execution from any done by others, it has given me an unusual standpoint from which to view certain problems which are both chemical and biological, and it is my intention to present in this paper some of the conceptions which I reached concerning the structure and function of the living cell. In doing this I wish it plainly understood that, so far as the theory is concerned, I am speaking tentatively and with the light now before me, reserving the privilege of making any alterations in my conceptions which may be justified or demanded by future investigations. With this reservation and recognizing that my time and your patience are not inexhaustible, I will at once plunge *in medias res*.

The Composition of the Living Cell.—When matter becomes endowed with life, it does not cease to be matter; it does not lose its inherent properties; it is not released

from the laws that govern its structure, its attractions and its motions. In studying the organized cell of living things, whether vegetable or animal, it should always be borne in mind that it is material in composition and subject to the fundamental laws that govern matter and possessed of those properties essential to matter. In order that this point, so essential to a proper understanding of the subject, should be thoroughly appreciated, it may be well to review some of the properties of matter as taught by the most advanced science of the day.

Tait says: "Matter is that which can be perceived by the senses, or is that which can be acted upon by or can exert force." Since force is the result of motion, we may say that anything and everything that moves or can be moved, or whose position in space may be changed, is matter. There are many forms of matter that can neither be seen nor felt, and can be recognized only by their motions.

Matter is indestructible; it may be successively solid, liquid and gas, but in passing through these phases it neither loses nor gains. It has always been and it always will be. It is without beginning and will be without end. Matter consists of infinitely small particles, called atoms. According to the computation of Lord Kelvin, the diameter of an atom is not greater than 1-50,000,000 of an inch; however, all atoms are not of the same size and weight. When like atoms combine they form chemical elements, of which about seventy are known. When unlike atoms combine, chemical compounds are formed and the number of these is beyond computation. Until the discovery and study of radium it was supposed that one element is neve converted into another, and consequently that the number of kinds of atoms is fixed and unchangeable. However, the evolution of helium from radium apparently demonstrates the formation of one element from another and it is within the range of sanity to suppose that all the elements

have been developed from a primordial ancestor, probably from the universal ether which pervades all space. Nothing has been created; everything has grown. Even silver, ron and other metals come into existence by being cast off from some ancestral element. It will be seen from this that even atoms do not represent the ultimate divisions of matter. Indeed, there are reasons for believing that the hydrogen atom consists of a nucleus about which some 700 particles or electrons revolve, and an atom of mercury is believed to consist of not less than 100,000 electrons. Atoms and electrons are in constant motion, and so small are they that the distances between them may be relatively as great as those between the planets of the solar system. The living cell is composed of molecules, made up of atoms, composed of electrons that are in constant systematic motion and may be compared to a group of stars with attendant suns, each of which is surrounded by its own planets.

Another property of matter is that it is gravitative. Every particle of matter attracts every other particle. When this attraction is manifest between masses it is called gravitation; between molecules it is called cohesion or adhesion, as the molecules held together are alike or unlike; between atoms it is known as chemical affinity or chemism.

Still another property of matter is inertia, by which term we indicate the inability of matter to change either its rate or direction of motion without being acted upon by other matter. It is of importance that this property of matter be held in mind in the study of cellular chemistry, and the proper mental picture of a cell molecule represents each of the atoms in the molecule, and each electron in each atom moving, each about its centre at a definite rate. Within the living cell molecule, change in number, kind and arrangement of atoms is constant; and changes in the direction and rate of the motion of the atoms are also susceptible to the influence of other matter and are of constant

occurrence. Whole groups of atoms are physiologically being dropped from the cellular molecule and being replaced by other groups split off from the pabulum upon which the cell feeds. In this way the cell renews itself and keeps itself supplied with energy.

Some eminent physicists are inclined to the belief that matter is made up of electric charges, but recognize that this is not a demonstrated fact as yet, and speak with caution. Lodge says: "There *may* possibly be two different kinds of inertia, which exactly simulate each other, one electrical and the other material; and those who hold this as a reasonable possibility are careful to speak of electrons as 'corpuscles,' meaning charged particles of matter of extremely small size, much smaller than an atom, consisting of a definite electric charge and an unknown material nucleus; which nucleus as they recognize, but have not yet finally proved, may quite possibly be Zero."

Cell Metabolism.—The only essential and constant difference between living and non-living matter is that within the molecules of the former there is constant metabolism, while in the latter no such process occurs. We are to conceive of the living molecule as made up of numerous atoms and each atom surrounded by its electrons; atoms and electrons are in ceaseless motion, groups of atoms are being constantly cast out of the molecule and replaced by new groups split off from matter outside the molecule. As soon as a molecule becomes the seat of assimilation and excretion it is no longer dead; it lives. As a result of assimilation it acquires the property of building up its own tissue; then polymerization follows and reproduction in its simplest form begins. The one phenomenon always manifested by living matter, and never exhibited by non-living matter, is metabolism. Verworn says: "Vital motion, metabolism, is a complex motion very strongly characterizing the living organism; it consists in the continual self-decomposition

of living substance, the giving off to the outside of the decomposition products, and, in return, the taking in from the outside of certain substances which give to the organism the material with which to regenerate itself and grow by the formation of similar groups of atoms, *i. e.*, by polymerization. This is characteristic of all living substance."

I have thought to give you some *new* conceptions of the living cell, and yet it must be admitted that Aristotle apparently recognized that metabolism is the one characteristic of living matter. He said: "Life is the assemblage of the operations of nutrition, growth and destruction." This Greek philosopher did not know about cells, molecules, atoms and electrons what is now known, but it must be acknowledged that he had a fairly clear conception of the most essential characteristics of living matter. Herbert Spencer has given three definitions of life and either may be applied to the conception which I am attempting to present. The first is: "Life is the coördination of actions." Coördination between assimilation and excretion is certainly essential to life, and failure of this coördination leads to death. The second is probably the best definition of life ever given, and fits our conception perfectly. It is: "Life is the definite combination of heterogeneous changes, both simultaneous and successive, in correspondence with external coexistences and sequences." The third is practically the same as the second, expressed in simpler terms, but in my opinion not so satisfactorily. It reads: "Life is the continuous adjustment of internal relations to external relations." Matter is alive when it feeds and excretes. Crystals grow and in a sense they multiply, but their growth is not intramolecular; it is by accretion. The living molecule not only absorbs, it assimilates. It chemically alters what it absorbs. The atomic groups taken into living molecules enter into new combinations. The living molecule is not stable, but is highly labile. Its composition is never con-

stant and it is never in a condition of equilibrium. There is a constant reaction between the living molecule and other molecules. Apart from other matter it could not exist. There is a constant interchange of atoms between it and other molecules. This is best seen in seeds, spores and ova. Matter existing in this form may be awakened into activity by proper stimuli; active life begins with the interchange of atomic groups, or with metabolism.

Why is there this constant atomic group interchange between the living molecule and outside matter? It is for the purpose of supplying the living molecule with energy. Allen has so ably expressed this fact that I make the following quotations: "The most prominent and perhaps most fundamental phenomenon of life is what may be described as the *energy traffic* or the function of *trading in energy*. The chief physical function of living matter seems to consist in absorbing energy, storing it in a higher potential state, and afterwards partially expending it in the kinetic or active form. We find in living matter a peculiar proneness to change its composition under the stimulus of slight changes in the energy-equilibrium between itself and its surroundings, energy being readily absorbed and readily dispersed. The absorption of energy coincides with deoxidation and the building up of large molecules; conversely the dispersion of energy coincides with oxidation and the disruption of the large molecules. The building up of these large molecules is always accomplished by slow steps; but when formed, the said molecules are very unstable, irritable, or, in modern phrase, *labile*. They may break down by degrees in some instances; in others their structure may be so precarious as to collapse on the slightest disturbance."

"The lability of such a molecule may be compared to that of a house of cards, which can be taken to pieces card by card, or may collapse at once. But the word lability

is applied, not only to *de*-structive, but also to *con*-structive instability. The molecules of living substance are prone to constructive as well as destructive changes; but as in the house of cards, the constructive changes are the most gradual; and as the structure grows more complex, construction becomes more difficult, and collapse is more imminent. It should be distinctly understood, however, that it is not the mere size of the molecules that makes them labile, but rather the manner in which they are linked together, and the amount of potential energy which is included in the molecule."

It is probable that in the absorption of energy by the living molecule oxygen is relieved from its combination with carbon or hydrogen and is attached to nitrogen, while in the liberation of energy the reverse takes place. Nitrogen and phosphorus, sometimes with iron and manganese, seem to be, as it were, the master elements within the living molecule. It is by virtue of their chemism that groups are torn from extra-cellular matter, taken into the living molecule and assimilated by an atomic rearrangement; and furthermore, it is on account of the lability of the compound thus formed that potential energy is converted into kinetic and cell work is accomplished.

That life resides within the molecule and that metabolic processes are intramolecular are shown by numerous investigations, some of the most important of which may be briefly stated as follows:

1. So long ago as 1867 it was shown by Hermann in his studies on the metabolism of isolated muscle that the carbonic and lactic acids produced in muscular contraction result from the action of intramolecular or combined oxygen. This was demonstrated by the observation that when a muscle is freed from all its uncombined oxygen under an air pump and then caused to contract in an oxygen-free medium it gives off carbonic and lactic acids. Contraction,

a vital muscle phenomenon, is thus shown to result from intramolecular changes.

2. In 1875, Pflüger kept a frog at a few degrees above zero in an atmosphere free from oxygen for twenty-five hours, and found that during that time the animal continued to give off carbonic acid and this could be produced under these conditions only through intramolecular changes. From these studies Pflüger concluded that the living content of the organism consists of proteid, which he designates "living proteid," in contradistinction to dead proteid, and the carbonic acid gas results from the decomposition of a labile proteid molecule, the nitrogenous constituents of which are capable, with the help of the fats and carbohydrates of the food, to regenerate "the living proteid molecule."

3. Recent research in my own laboratory has shown that both the toxic and carbohydrate groups of the cell of the colon bacillus are held in chemical combination with other constitutents of the cell. This microorganism will grow in a medium which contains organic nitrogen as amino compound only and with this nitrogen and inorganic salt as its sole food, it builds up by synthetical process a complex glyco-nucleo-proteid, forming a large molecule which contains as atomic groups, pentose, nuclein bases, amino and diamino compounds. These constituents are held chemically in the cell; they are constituent groups of the large cell molecule. They cannot be washed out by physical solvents, and can be isolated only by chemically breaking up the cell molecule.

Besides the above mentioned experimental data showing that cell life manifests itself by intramolecular reaction the following general considerations indicate the same thing:

(*a*) In taking its food the cell, whether it be vegetable or animal, whether it be that of a unicellular or that of a multicellular organism, manifests a selective action which

can be best explained—indeed, I might say, can only be explained—on the ground that it is due to mass chemical affinity. Mass attraction is, so far as I know, never specific; molecular attraction is specific only in general terms and not in the sense of forming synthetical compounds; but atomic attraction, or chemical affinity as it is usually designated, is specific, or at least selective. This fact, as is well known, is the basis of the side chain theory of Ehrlich, who, upon this principle, explains the nutrition of cells, the action of many therapeutical agents and the production and action of antitoxins. It is well known that certain poisons have a selective action on certain tissues, and this means that the chemical affinity between the poison and the constituents of certain cells is greater than that between this poison and other cells. If pharmacology and toxicology ever become exact sciences it will be, most probably, through investigations directed along this line.

(*b*) The fact that the secretions of cells are specific is a strong argument for the theory that action on the pabulum upon which they feed is intramolecular. The liver cells produce bile pigments and acids, each of the digestive fluids elaborates its specific products, the specific secretions of the adrenals and the thyroid gland have been studied and are now largely and successfully employed therapeutically. And still all these organs are supplied with the same blood and lymph. Certainly, the only possible explanation for these well established facts is that of a chemical reaction, or an intramolecular interchange, between the cells and the constituents of the substances with which they are brought into contact.

While other arguments might be adduced to show that metabolic processes, the only phenomena with which we are acquainted that are characteristic of all living matter and which do not occur in dead matter, are due to intramolecular reactions, it seems to me that those already given are sufficient to establish my thesis *i. e.*, life is molecular.

If I have made good my contention so far, it follows that life begins with the capacity of growth and reproduction. The life of such a molecule depends upon its continued reaction with matter outside of itself, or, in other words, it must feed; and reproduction in its simplest form results from polymerization. In this way the wonderful experiments of Loeb upon the artificial fertilization of certain ova may possibly be explained. The ovum is not alive; it possesses only latent life and when acted upon by certain stimuli it begins active life. The stimulus may be a spermatozoon or some inorganic salt in a certain definite strength of solution.

If life be molecular, it is possible that its lowest manifestations are without form. They may be infinitely small, and it is not beyond the range of possibility that they may exist in either or all the three known conditions of matter, solid, liquid, or gas.

Cellular life is the only form of life that we know at present, but the statement "omne vivum e cellula" is dogmatic and probably is not true. It is too early yet to predict what influence more exact studies of viruses that pass through porcelain filters will have upon this dictum.

The Cell not the Unit of Life.—The following quotation from Nussbaum, as given by Loeb, shows that the biologist recognizes that the cell is not the unit of life: "The cell is not the ultimate physiologic unit, even though it must remain such for the morphologist. We are, however, not able to tell how far the divisibility of a cell goes, and how we can determine the limit theoretically. Yet, for the present it will be well not to apply to living matter the conceptions of atoms and molecules, which are well defined in physical chemistry. The notion, introduced by Nægele, might also lead to difficulties, as the properties of living matter are based upon both nuclein and protoplasm. * * * The cell, consequently, represents a multiple of individuals."

Pflüger has shown that the egg, which has been thought to be a biologic unit, can give rise to many individuals, and Loeb states that his own experiments, as well as those of Driesch, confirm this finding.

It is highly probable that the lowest forms of life cannot feed upon proteids. This is true of the yeast cell. These cells grow rapidly when placed in a solution of sugar and nitrates, but proteids must be broken up by putrefactive bacteria before the yeast organisms can feed upon them. Indeed, many of the cells in the body of man cannot feed upon complex proteids, which must be split up by the digestive enzymes into much smaller and simpler groups before the cell molecules can feed upon them. Even the carbohydrate, starch, must be hydrated and its elements rearranged as constituents of a more labile molecule before it can become a source of energy in muscle. Proteid solutions injected into the blood of man are poisonous, but the same substance, after being properly split up, is an essential cell food.

Some light has been thrown upon the chemistry of the growth of the cells of germinating seeds by the researches of E. Schulze and his students. As is known, the seed consists of the germinating cell and the stored up food material. The cell in a lupine seed is not actively alive; it possesses only latent life. When placed under conditions favorable to the development of active life, a ferment begins to break up the proteid food material stored in the seed and as a result of the chemical cleavage induced by the ferment relatively simple nitrogenous bodies, such as the amino acids, tyrosin and leucin, and the diamino acids, arginin and lysin, are formed. These bodies serve as food material for the germinating cell and latent life is quickened into the active form and growth begins.

If the characteristic phenomena of life are due to intramolecular reactions, we must conceive of the living cell,

whether it belong high or low in the scale of development, as consisting in its essential or vital part of a chemical compound made up of complex molecules, composed of atoms, each surrounded by its electrons, all in motion and with a constant, probably a rythmical, absorption of atomic groups from other molecules, and with a like constant discharge of atomic groups.

This living molecule feeds by splitting off such groups as it may need from the pabulum within its reach, or it may absorb entire molecules, at the same time rearranging the atoms and making them a part of itself. When in ordinary physiological function, a portion of this molecule, which we may designate its chemical nucleus, remains undisturbed and regenerates the whole, supplying its waste by the absorption of new material. Cellular assimilation consists in properly locating the recently acquired atomic groups within the molecule.

Certain cell molecules, under proper stimuli, rearrange their atomic grouping, polymerize and thus multiply. This multiplication may be physiological or pathological. Rapid proliferation may tend to inability to function or to react with the food supply, and consequently destroy the molecule or lead to the death of the cell.

In his very interesting monograph on the "Biogen Hypothesis," Verworn objects to saying that a molecule lives. He states that it is illogical. "A living thing is only that which demonstrates the phenomenon of life,—something that changes itself. A molecule of a given compound, so long as it remains unchanged, cannot be said to be living." Then, in order not to speak of living molecules, he introduces the term "biogen molecule." Surely, this is a distinction without a difference. I certainly agree with the distinguished German physiologist that a molecule of a cell, *so long as it remains unchanged*, cannot be said to be living, but the point is that living molecules do not remain

unchanged. When life is latent, as it is in seeds and spores, the molecules cannot be said to be alive; but when placed under suitable conditions, then the change between atomic groups in the molecule and the external food substance begins, and life first manifests itself. However, it matters but little, I suppose, whether we speak of living molecules or designate them as "biogen molecules."

Cell Secretions.—With the conception of a living cell that I have attempted to present, it must be evident that its secretions consist of the atomic groups cast out as a result of its metabolism, and as the cells of different organs are unlike in their chemical composition, it follows that the secretions are specific. Outside the body hemoglobin breaks up, or may be broken up chemically, into hematin and a globulin. In this case the colored split product contains the iron; but the liver cells produce from hemoglobin bilirubin and an iron-containing proteid. In these two reactions the line of cleavage is quite different. The secretions of some cells enter into a more or less energetic reaction with certain extra-cellular compounds with which they come in contact. This is true of the digestive enzymes. Other secretions apparently are made for the purpose of reacting with or at least affecting the reactions of the molecules of other cells. This seems to be true of some, at least, of the so-called internal secretions, such as those of the thyroid and adrenals.

Ferments.—A most important group of cellular secretions consists of the ferments or enzymes. Without going into the history of the theories that have been advanced concerning the nature of these bodies, it seems to me that we are no longer justified in speaking of "organized and unorganized" ferments. All the ferments are cellular secretions. The work of Buchner on the ferment of the yeast plant seems to be positively convincing on this point. Oppenheimer has defined a ferment in a manner quite in

accord with the latest and best experimental investigation. His definition is as follows: "A ferment is a catalytically acting substance which is produced by living cells, to which it is more or less firmly bound, whilst it is not associated with the vital processes of the cells (which produce it); ferments are capable of inaugurating chemical processes which take place spontaneously (without the presence of the ferments), but proceed more slowly. In this process the ferment itself remains unchanged. Ferment action is specific, *i. e.*, each ferment manifests its activity only on substances of certain structural and stereo-chemical arrangement."

I am conscious that my translation of this definition is not altogether satisfactory, and, in order to give a more exact interpretation of it, as I understand it, I offer the following explanatory statements:

1. Every ferment is a cellular product; it is a cellular secretion; a substance of definite chemical composition formed by the rearrangement of the atomic groups within the cellular molecule.

2. The action of the ferment, while it is determined by the cell which produces it, is not concerned in the "energy traffic" constantly going on between the molecules of the cell which produced it and other molecules external to this cell. With our present limited knowledge of the chemistry of the cell molecule it is impossible, in many cases at least, to distinguish between the chemical reactions resulting from cell metabolism and those due to ferments. I am inclined to the opinion that more exact knowledge will show that the autolytic changes that take place in many cells after death, and which have furnished the theme of so many papers recently, will be found not to be due to ferments at all, but to the cessation of metabolic reaction and consequent dissociation of the constituent groups of the cell molecules.

3. The function of a ferment is to hasten chemical reactions which take place, but much more slowly, without the presence of the ferment. It seems to me that a clear conception of this point gives one a key to the action of ferments in general. I have already in this paper called attention to the fact that inertia is a universal property of matter; that the direction and rate of movement in matter cannot be altered spontaneously. A ferment is a substance of cellular origin which by its presence changes the *tempo* of chemical reaction. I borrow this word "tempo" from the musician because it expresses my meaning better than any other word in my vocabulary. I am fully aware that this does not explain *why* the ferment acts by its presence, but it is worth much to have a conception of *how* it acts, provided, of course, that this conception be correct. Furthermore, it must be admitted that the *modus operandi* of ferments is still beyond our ken. Some think that certain atoms or atomic groups are detached from one of the substances, combine with the ferment, and then are passed on to the other substances. On this supposition the ferment does enter into the reaction, but is constantly regenerated. Others hold that the ferment combines with the fermentable substance, making its molecule so labile that it falls to pieces and that in the dissociation the ferment is again set free. There are weighty objections to either of these theories, but time will not permit me to state them in this paper, which on this point at least is intended to be suggestive rather than exhaustive.

Part II.—The Bacterial Cell.

Preparation of Bacterial Cell Substance.—Bacteria of various species, both pathogenic and non-pathogenic, have been grown on the tanks. When the growth has reached its maximum, the time required for this varying with the species and the temperature, the growth is de-

tached from the subjacent agar with sterilized bent glass rods and drawn into sterilized flasks with the aid of a water pump. The *cellular* substance is then washed with dilute, and later with stronger, alcohol. After thorough extraction with this agent it is placed in Soxhlets and thoroughly extracted with ether; next it is rubbed up first in porcelain, then in agate, mortars and passed through fine meshed sieves. This treatment yields fine, impalpable powders, the color of which varies with the germ, but is generally white because the bacterial pigments are soluble in alcohol.* These powders, when examined microscopically, show the cells, broken more or less, generally but little, by the rubbing to which they have been subjected. They still take the stains to which the original cultures respond with some variations with certain species. In this way we have obtained the cellular substance of the following microorganisms: b. prodigiosus, b. violaceus, s. lutea, s. aurantiaca, b. coli communis, b. typhosus, b. pyocyaneus, b. anthracis, b. tuberculosis, b. diphtheriæ and m. pneumoniæ.

The Toxcity of Bacterial Cell Substance.—The cell substance obtained from each of the above mentioned microorganisms has been found to be toxic and its toxcity has been studied and reported upon by my students, Detwiler, Wheeler, Leach, Gelston, Marshall, J. W. Vaughan, and V. C. Vaughan, Jr. It is interesting to note that when injected intraperitoneally in animals, the cell substance of the b. prodigiosus is more potent than that of any of the pathogenic bacteria, being more than fifty times as active as the cell substance of b. anthracis; and even the lemon sarcine, the least toxic of the non-pathogenic organisms examined, surpasses the anthrax bacillus in the potency of its intracellular poison. This shows, as I have elsewhere

* A preliminary report was made on certain bacterial pigments by my student, Detwiler, in 1902. (Transactions of the Association of American Physicians.)

stated, that the prodigiosus is non-pathogenic to the higher animals, not from its inability to elaborate a poison, but because it cannot grow and multiply in the animal body; while on the other hand, the anthrax bacillus is highly infectious to some of the higher animals, not from the intensity of the poison which it elaborates, but rather from the fact that in these animals this bacterium finds the conditions favorable for its growth and multiplication. In a more general way this statement may be made as follows: a non-pathogenic bacterium may be capable of elaborating a highly active poison in artificial cultures or from dead material, but its chemism does not enable it to split up and feed upon the constituent tissues of the living animal.

The more finely bacterial cell substance is divided the more potent is it in its action. This was shown conclusively by the studies of Marshall and Gelston and is, in my opinion, due to the more ready solubility of the finely divided powders in the fluids of the body; the greater the relative surface of the germ surface exposed to the action of both the formed and the soluble constituents of the animal tissue, the more speedily is the poison set free and the more certain is death.

It may be interesting to give somewhat more in detail a statement of the amounts of cellular substance obtained from different bacteria found necessary to produce fatal results when injected intraperitoneally in guinea pigs. These amounts vary with the degree to which foreign matter is removed from the cell substance, the fineness to which the powder has been reduced and the length of time and the conditions under which the material has been kept. When frequently exposed to air and light the cellular substance decreases in toxcity, at times quite appreciably and suddenly. This I believe to be due to a hydration of the toxic group of the cell molecule and its consequent conversion into an inert or at least a less poisonous body. It is highly

probable that this occurs to a greater or less extent in cultures in which individual organisms must die from time to time and whose cells probably undergo autolytic changes. The following figures gives the average fatal amounts of the freshly prepared, finely dried cellular substance of some of the bacteria with which we have worked:

Table 1.—The toxcity of the finely powdered cellular substance when injected intraperitoneally in guinea pigs.

Name of Organism.	*Proportion of cell substance to body weight.*
Bacillus anthracis	1:1700
Sarcina lutea	1:2050
Micrococcus pneumoniæ	1:10000
Sarcina aurantiaca	1:25500
Bacillus violaceus	1:26500
Bacillus diphtheriæ	1:33000
Bacillus typhosus	1:40000
Bacillus pyocyaneus	1:50000
Bacillus coli	1:75000
Bacillus prodigiosus	1:90000

When suspended in water bacterial cell substance may be boiled without destroying its activity. Marshall and Gelston found that heating the cell substance of b. coli communis to 134° for 15 minutes did not appreciably lessen its toxcity, and Cooley and I heated the same substance in a sealed tube to 164° without rendering it inert. In a paper before the Association of American Physicians in 1901 I made the following statements concerning the toxcity of the colon cell:

1. The toxin is contained within the cell, from which it does not, at least under ordinary conditions, diffuse into the culture medium.

2. The toxin is not extracted from the cell by alcohol or ether.

3. Very dilute alkalis do not extract the toxin from the unbroken cells.

4. The unbroken germs may be heated to a high temperature with water without destruction of their toxin.

5. Heating the cell substance for an hour at the temperature of the water bath with water containing from 1 to 5 per cent. hydrocholric acid breaks up the cell content and lessens, but does not destroy, the toxicity of the cell content. Prolonged heating may render the toxin inert.

The Chemistry of the Bacterial Cell.—With the method employed and previously described, everything soluble in water, in dilute salt solution, in alcohol and in ether is removed from the cell substance. A discussion of the extractives obtained by the above mentioned solvents must be postponed and will, I hope, be presented to the profession at some future time. These extractives contain inorganic salts, fats, wax, traces of carbohydrates and several proteid bodies. These substances belong in part to the culture medium, certain constituents of which become more or less intimately adherent to the cells without being assimilated by them; others consist of split products formed by the action of the living cells upon the constituents of the culture medium; others still are secretions of the living cell, representing atomic groups dropped from the living molecule in the process of metabolism and in the traffic in energy constantly carried on between the living, growing and multiplying cell and the outside matter which constitutes its pabulum; and finally there are substances that result from the autolysis of the dead cells and some of these are identical with the bodies obtained by splitting up the cell molecules by chemical means and which will be described later. With some species of bacteria the important bodies that result from chemically breaking up the cell molecule can be best obtained by permitting the process of dissociation, which occurs sooner or later in all cells after death, to proceed until the greater part of the cell substance passes into the soluble form. When this happens the separation of the toxic from the non-toxic groups becomes easy of execution. There is much valuable information to be obtained

by the study of these extractives, but I have not done enough with them to enable me to make many positive statements at present. Moreover, it is the purpose of this paper to give the results of the studies of my students and myself on the cell molecule or that portion of the cell that remains after everything that can be washed out with physical solvents has been removed, and when I speak of the cell as being in its essential part a chemical compound of definite composition and structure it must be held in mind that I refer to that part of the cell that remains after the extractives have been removed. (Except of course, when autolysis has taken place.) I am anxious that all should clearly understand and fully comprehend this point.

The essential part of the cell, that which remains after the removal of the extractives, can be split up by chemical agents and these split products will now be discussed.

The toxic group of the bacterial cell molecule.—When bacterial cell substance, freed from extractives and prepared after the method already given, is heated in a flask with a reflux condenser with from 15 to 25 times its weight of a 2 per cent. solution of sodium hydroxid in absolute alcohol, the cell molecule is split into toxic and non-toxic groups. The poisonous portion is now in solution in the alcohol and in case of b. coli and b. typhosus it constitutes about one-third by weight of the cell substance. For the complete solution of the poisonous group three extractions at 78° for one hour each have been found to be sufficient. It is essential that the alcohol used in the extraction should be absolute; if it contains water, hydration proceeds too far, much of the toxic body is destroyed and it will be found on removal of the alcohol that a sticky, gummy mass, which it is quite impossible to dry, remains. The alcoholic solution of the toxic part is neutralized with hydrochloric acid, avoiding an excess, and the precipitated sodium chlorid removed by filtration. The alcoholic filtrate is evaporated in vacuo

below 40°, the residue redissolved in absolute alcohol, and this may be repeated several times. When the sodium chlorid has been removed the toxic part of the germ substance is obtained in a brownish mass which may be ground into a fine powder and weighed.

By the above mentioned treatment the cellular substance is broken into two portions, one of which is soluble in absolute alcohol and is poisonous, while the other is non-soluble in alcohol and non-poisonous. The dead cellular substance I have designated as the "crude bacterial poison," while the part of this rendered soluble in alcohol by treatment with an alcoholic solution of alkali I have called the "crude soluble poison."

The toxic part of the bacterial cell is soluble in water, ethyl and methyl alcohol, and insoluble in ether, chloroform, benzine and petroleum ether. It is more readily and freely soluble in absolute alcohol than in water, which is unusual in a proteid body. When a solution of it in absolute alcohol is evaporated in vacuo it forms a brownish hydroscopic powder which dissolves in water forming an opalescent, acid solution. The opalescence may be largely or altogether removed by filtration through hard paper. When neutralized with sodium bicarbonate, a brownish, non-toxic substance is precipitated and may be removed by filtration.

The crude soluble toxin in aqueous solution gives all the proteid color reactions with the exception of that of Molisch. It gives the Millon test beautifully and in very dilute solution. It does not reduce Fehling's solution, either directly or after boiling with dilute mineral acid. It contains sulphur, but not that sulphur group that is split off from most proteids on boiling with caustic alkali. Aqueous solutions are not coagulated by heat, but a precipitate is produced on the addition of mineral acid. The heavy metals, as mercury and copper, produce voluminous precipitates. Saturation of an aqueous solution with ammonium sulphate

throws down a sticky resinous portion and when this is removed by filtration, both the precipitate and the filtrate give the proteid reactions. Platinum chlorid produces a precipitate in both alcoholic and aqueous solutions of the "crude, soluble toxin" and the filtrate, after removal of the platinum, has but little or no poisonous action; but the platinum precipitate, which contains the whole, or the greater part, of the poison refuses to dissolve in any of the agents which we have tried and, consequently, we have been able to reach no satisfactory method of purifying the poison. Separation into two portions by saturation with ammonium sulphate is not sharp and distinct; sometimes the filtrate is more, again less poisonous than the precipitate, although the weight of evidence is in favor of the precipitation of the poisonous portion by ammonium sulphate added to saturation. Undoubtedly there are conditions influencing this separation which up to the present time have escaped our knowledge.

As to how to classify this poisonous bacterial substance we must admit that we are still very much in the dark. The fact that the "crude soluble toxin" gives all the proteid color reactions with the exception of that of Molisch leads us to say that it is a proteid body, while against this view stands its ready solubility in absolute alcohol. The precipitation of a portion of the "crude soluble toxin" by saturation with ammonium sulphate while both precipitate and filtrate, after removal of the salt, give all the proteid color reactions, with the exception of that of Molisch, leads us to say, for the present and until continued work increases our knowledge, that the poisonous group in the bacterial cell molecule consists of a bacterial albumose and that this differs from the better known vegetable and animal albumoses in its ready solubility in absolute alcohol. It seems probable that the albumose is the poisonous body and that it is easily converted into a pepton-like body which is either inert or

has but little poisonous effect upon animals. It may be that the real poison is mixed with this proteid body, while chemically it is no part of it, but we have tried all known methods of extracting a poison from this proteid and having failed in all these attempts we must for the present assume that the proteid itself is the poison. Indeed, we started upon this research and adopted the method that we have followed on the theory that the poison in the bacterial cell is neurin and that it loses its poisonous properties by easy conversion into cholin. We were led to suspect this on account of the similarity between the poisonous action of neurin and that of the active body in the bacterial cell. If this could be proven it would afford a ready and rational explanation of the injurious effect of the bacterial cell on the animal body; but we have labored earnestly now for some years to find neurin in the bacterial cell with constantly negative results. We do not say that a neurin group does not exist in the bacterial cell; indeed we still believe that the poisonous agent in the bacterial cell is physiologically at least a neurin, but we are compelled to admit that we have not been able to prove the presence of either neurin or cholin chemically.

The "crude soluble toxin" is an acid body with sufficient acidity to slowly decompose the alkaline bicarbonates forming salts which act like the free acid, but more tardily.

The Action of the Colon Bacillus and its Poisonous Group on Animals.—It is interesting to study the effects of the living colon bacillus, the dead bacterial substance and the soluble poison split off from the bacterial cell on animals.

We have used a colon bacillus 1 c.c. of a 12 hour or older culture of which has invariably killed guinea-pigs, when injected intraperitoneally, within 24 hours. When only 1 c.c. is given the animal shows no effects for from 10 to 12 hours; when a larger quantity is given the symptoms

appear earlier. This period of incubation represents the time necessary for the bacillus to multiply and be destroyed or broken up sufficiently to liberate enough of the poison to induce observable effects on the animal. In reality this is the critical period of the infection and the result depends upon whether or not all the bacteria are destroyed before they have multiplied sufficiently to furnish a fatal dose of the poison. "It is during this period that individual resistance and acquired immunity are important factors acting by causing increased bacteriolysis and the destruction of all germs before a fatal dose of poison has been set free. During this time the temperature of the animal may rise to a greater or less extent or may remain stationary; the animal remains active, eats; its coat is not roughened and it appears in all respects as well as a normal animal. At the end of this period, however, the appearance changes. The animal becomes less active. It remains in one corner of its

CHART I.

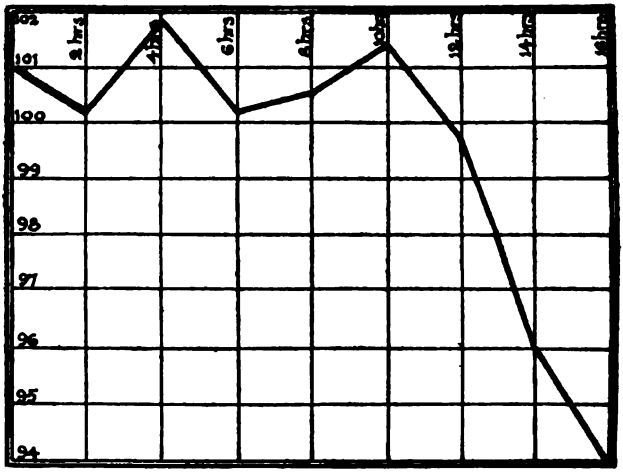


CHART I.—Temperature curve of guinea-pig after inoculation with 1 cc., 16 hr. bouillon culture of the colon bacillus. Death occurred 20 hours after inoculation.

cage; its coat becomes roughened; it hangs its head and apparently enters into a state of stupor. At the same time the rectal temperature begins to fall abruptly, as can be seen from a study of Chart I.

Indeed, this fall of body temperature is often the first marked symptom, and when occurring to a marked degree it is invariably a bad omen. The body temperature will often fall from 101° F. to 94° F. or even lower within from 2 to 4 hours, and this fall is progressive and continuous until the animal's death, immediately preceding which a temperature as low as 87° F. or 86° F. is not uncommon. At the same time the animal shows signs of the most marked peritoneal inflammation, as is evidenced by rigidity and spasm of the abdominal muscles on pressure. At autopsy, the only gross lesion present is a marked hemorrhagic peritonitis with a large amount of bloody fluid containing intact red corpuscles and leucocytes in the peritoneal cavity. The parietal and visceral peritoneum are studded with minute punctiform hemorrhages. Hemorrhage is an especially prominent feature in the great omentum, and is present to a less marked degree in the mesentery."

With the dead bacterial substance, or crude toxin as I have called it, the symptoms in the animal and the post mortem findings are exactly the same as when the living culture is used. The only difference is in the period of incubation. Of course the final issue when the dead germ is used depends upon the amount injected, since there is no possibility for increase in the poison after injection. The animal remains apparently well for about four hours and then its temperature begins to fall abruptly as is shown in Chart II, in which case the dose given was not sufficient to cause death.

When the "crude soluble toxin" is administered the period of incubation and the time necessary for the splitting up of the cellular substance are both done away with and

CHART II.

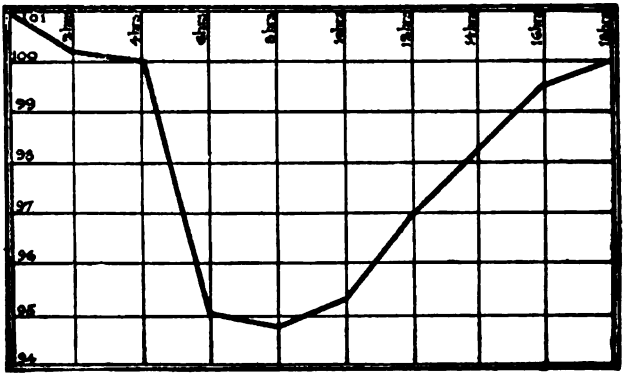


CHART II.—Temperature curve of guinea-pig after intraperitoneal injection of non-fatal dose of crude bacterial cell substance.

the fall of temperature begins within 15 or 20 minutes as is shown in Chart III. If the dose be a fatal one the fall continues until death; if it be less, the first indication of improvement is an upward move in the temperature.

CHART III.

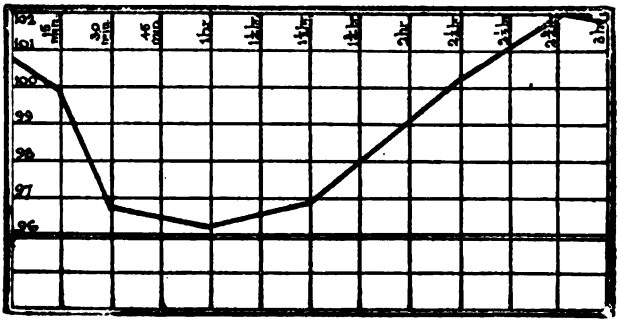


CHART III.—Temperature curve of guinea-pig treated with 45 mgs. of the soluble poison intraperitoneally.

The hemorrhagic peritonitis induced by both the living and the dead bacterial cells is wholly wanting when death results from the soluble poison. This seems to me quite

interesting and I offer the following explanation: This inflammatory condition manifesting itself in a severe and extensive hemorrhagic peritonitis is not due to any activity on the part of the living bacillus, because, as we have seen, it is just as marked when the unbroken dead germ is thrown into the peritoneal cavity as it is when a living culture is employed. For the same reason I must conclude that this inflammatory condition is not caused by, nor is it even connected with the growth and multiplication of the invading microorganism, but it is a result of the disintegration and destruction of the bacteria. It is in my opinion due to the chemotatic force acting between the constituents of the bacterial cells and constituents of the cells of the animal body, and this force is of sufficient intensity to break up both the animal and the bacterial cells. In other words, I believe that bacterial inflammation is essentially a chemical process or is due to the disruption of cell molecules through the chemical affinity between certain groups in the bacterial cell and certain groups in the cells of the animal. So long as the bacterial cells are alive the chemism that holds the living molecule together tends to resist this process of disintegration. I have attempted to investigate this point by placing the dead bacterial cell substance in collodion sacs in the abdominal cavity of rabbits, but in every instance, in which this experiment has been made, the animals have died and after death a localized area of inflammation about the sac has been observed. This point, however, deserves more extended experimentation.

The soluble poison diffuses through collodion sacs and animal membranes, but it does so slowly and when many times the fatal dose, determined by intraperitoneal injection, is introduced into the stomach of a fasting rabbit, the animal does not die. This I suppose to be due to the slow absorption of the poison rather than to its destruction. When the dead bacterial cell substance is subjected to arti-

ficial gastric digestion there is no diminution of its toxicity, but the poison is apparently slowly destroyed by pancreatic digestion.

The soluble poison of the bacterial cell apparently kills by its effect upon the respiratory center. The heart continues to beat normally for some minutes after respiration ceases. In this respect the bacterial poison bears a close resemblance to neurin. The quantity of the soluble poison necessary to kill a guinea-pig depends upon the extent to which purification of the poison has been carried. Usually it contains considerable sodium chlorid, and this can be removed only by repeated evaporation and re-solution in absolute alcohol. The most potent poison that we have obtained killed guinea-pigs in doses of 8 mg. given intraperitoneally, while the poison with which we have made most of our experiments required 60 mg. The fatal dose intravenously is about one-sixth, and that required when given subcutaneously is about twice the quantity needed intraperitoneally.

Our method of extracting the active poison from the bacterial cell is by no means satisfactory, and much of the poison is destroyed in the process. For instance, a bacterial cell substance 5 mg. of which will kill a guinea-pig yields less than one-third of its weight of this "crude soluble toxin," and when the best results are reached not less than 8 mg. of this kills guinea-pigs. I am of the opinion that the poison exists in the bacterial cell as a polymer, and much of this is hydrolized into an inert, or a relatively inactive, form by our process of extraction. If the bacterial cell could be broken up outside the body as it is within the body, we should have a larger yield of the active substance. We have tried various other methods of extraction, but up to the present time we have failed to improve upon that already described. We have detached from the cell substance of numerous toxicogenic bacteria bodies very

similar in their physical and chemical properties and in their effects upon animals. The general clinical symptoms of many of the infectious diseases are markedly similar, and we distinguish one from the other more by the organs involved than from differences in the effects of the poison upon the individual. A typhoid septicemia can hardly be distinguished from one induced by a paracolon or paratyphoid organism, and, indeed, a general tuberculous septicemia bears so close a resemblance to typhoid that it may lead astray, for a time at least, even a skilled diagnostician. A meningitis may be caused by any one of a number of micro-organisms, and no one can determine absolutely from the symptoms the exact name of the invading organism. Moreover, it has been shown by experiments in my laboratory that the molecules of egg albumen and of pepton contain poisonous groups very similar to, if not identical with, those of the cells of certain toxicogenic bacteria.

If I may be permitted to draw some inferences from these studies on bacterial endotoxins, I will state them as follows: The invading bacterium grows and multiplies. The tempo in which these processes proceed is variable, depending upon certain properties inherent in the infecting cell and at present quite unknown to us, and also depending upon the condition of the food material, in the animal, utilizable by the infecting cell and the resistance offered to its growth by the cells of the infected animal. All of these things, and possibly others as well, influence infection. From my own work using different strains of the same organism on individual animals of the same species, I have concluded that the virulence of the bacterium is the most important factor in determining the time and certainty of the death of the animal, and that the virulence of the bacterium is measured by the tempo with which it multiplies in the animal body. For the study of this question the colon bacillus is, in my experience, poorly suited, because

with this organism there are not the wide variations in virulence that we find in certain other pathogenic bacteria. In my investigation of this subject I have employed different strains of the pneumococcus. Some of these are only feebly pathogenic or virulent, while others are most highly so. One strain requires at least 1c.c. of a culture 12 hours old to kill a guinea-pig, while with another strain one-millionth of a c.c. kills. But when two guinea-pigs have died from the effects of these cultures, one from the feebly and the other from the highly virulent culture, so far as one can judge from a close inspection of the organs of the dead animal and the distribution and the number of bacteria present, the growth of the infecting cells has reached the same point in the two animals, and what is more scientifically exact when these two strains are grown artificially and the poison extracted from the cells, like weights of the purified cell substance yield like amounts of the poison. From this I conclude that differences in virulence between different strains of the same pathogenic organism depend upon the tempo or rate with which the organ multiplies in the animal body. This may not be true of all pathogenic bacteria, but it seems to be true of the pneumococcus. I have been surprised at the tenacity with which some strains of the pneumococcus hold their virulence through many generations of artificial cultures.

There is another point in favor of the idea that the virulence of the bacterial cell is dependent upon its rate of growth in the animal. The colon culture with which I have done much of my work varies but little, if at all, in virulence; 1c.c. of a culture 12 hours old invariably kills guinea-pigs when injected intraabdominally; I have a pneumococcus that kills, as I have already stated, in one-millionth of a c.c., and yet 5 mg. of the dead bacterial cell substance of the colon bacillus kills, while it requires about 60 mg. of the dead bacterial cell substance of the pneumo-

coccus to kill. It may be that the latter is not freed from foreign matter so thoroughly as the former, but this can hardly explain so wide a difference, and I must for the present at least hold that virulence in bacteria is largely due to rate of growth in the animals, and this is due to some form of energy in the bacterial cell, the nature of which is, at present, quite unknown.

I think it highly probable that the cells of the infecting organism feed upon the soluble, or at least on the lifeless and unorganized constituents of the animal body, and that the lesions of the infectious diseases are not due directly to the multiplication of the invading bacteria, but to the poisons elaborated by these invaders. Prudden and others have shown that tubercular lesions may be induced by the injection of dead tubercle bacilli, and we have seen that the hemorrhagic peritonitis caused by the injection of the crude colon toxin is undistinguishable from that found after death from the living bacillus.

The pathogenic bacterium assimilates the nutritious constituents of the fluids of the animal body, builds them into its own tissue, converts them into substances foreign to the host, and finally when the bacterial cell goes to pieces either from spontaneous dissolution or through the aggressive action of some animal cell these reconstructed chemical groups are set free and poison the animal, inducing lesions in various tissues, and, in many instances, so interrupting the vital functions as to cause death.

The Non-Toxic Residue of the Bacterial Cell.— When the cellular substance of the colon or typhoid bacillus has been thoroughly extracted with a 2 per cent. solution of caustic alkali in absolute alcohol about two-thirds by weight of the material is left insoluble in alcohol and this we have designated as the "non-toxic residue" or simply as the "residue." This residue responds to all the proteid color reactions with the exception of the Millon test.

This reaction, which is given in great perfection by the poisonous portion of the bacterial cell and which follows the poisonous part in all attemps to purify it, fails with the residue after purification by repeated solution in water and precipitation with alcohol. If a given residue responds to the Millon test it is proof that the poisonous part has not been wholly removed as can be demonstrated on animals. This is not, however, true of all bacteria; the non-poisonous residue of the tubercle bacillus does respond to the Millon test and I can positively assert its absence only in the colon and typhoid residues. This indicates a chemical relationship between these organisms and a chemical difference between them and the tubercle bacillus. Another interesting fact is that the Molisch reaction, which fails wholly with the poisonous portion of the colon and typhoid organisms, is given abundantly and promptly by their residues. Furthermore, the Millon test is positive not only with the poisonous portions of the colon and typhoid bacilli, but also with this portion of all the pathogenic bacteria, including the tubercle bacillus, which we have studied, as well as with the poisonous group of egg albumin and pepton. This indicates a chemical relationship between the poisonous portions of all these bodies. The fact that the toxic part of colon and typhoid bacilli gives the Millon reaction promptly and perfectly, while it fails absolutely to respond to Molish test, and the residue of these bacilli give the Molish test promptly and perfectly, while it fails in the Millon test, is strong chemical evidence that our method of splitting up these bacterial cells gives natural cleavage products. We can distinguish between these split products by chemical tests as well as by their effects upon animals. In neither case have we obtained bodies chemically pure, but it is a satisfaction to know that we have made a chemical separation.

From the fact that the toxic portion of the colon cell

fails to give the Molisch test while the residue responds to this reaction promptly I infer that the carbohydrate group of the cell molecule is confined to the residue. There are other chemical differences between the poisonous and the non-poisonous portions of the colon bacillus. The former contains about 12 per cent. of nitrogen and the latter not more than 5 per cent.; while the poisonous part yields less than one per cent. of phosphorus and the non-poisonous part contains more than 3 per cent. of this element. The chemistry of both of these split products is now being investigated, and I hope to be able at some future time to make more exact statements on these points.

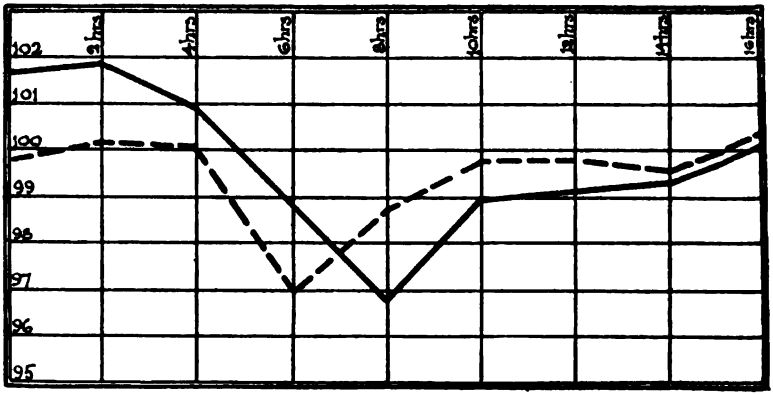
Part III.—The Production of Active Immunity with the Split Products of the Colon Bacillus.

Attempts to Establish Immunity with the Poisonous Portion.—The poisonous portion of the colon bacillus acts on animals so promptly and so powerfully that attempts to secure a tolerance to it frequently end in the death of the animal. If one rapidly increases the size of the successive doses this is certain to be the result. It may be found to be possible by combining the poison with an alkali and thus retarding its effect to secure better results, but this has not been systematically tried yet. Inasmuch as this subject has been discussed in some detail by one of my students in a recent paper* I will content myself with a brief condensation of his results. By proceeding carefully and gradually increasing the doses a point may be reached where the animal will bear treatment with from two to three times the amount that would surely kill an untreated animal. This indicates the development of either a slight degree of immunity or the establishment of a mild tolerance for the poison. Which of these actually results can be positively determined only after a satisfactory investiga-

* See bibliography at the close of this article.

tion concerning the production of an active antibody, and up to the present time our attempts along this line have not been followed by uniform results. Animals brought into this condition, whether it be one of increased tolererance or immunity, bear from two or four times the fatal quantity of living cultures of the colon bacillus. It is an interesting fact that animals that have had a single non-fatal dose of the poisonous portion are able to withstand twice the lethal dose of the living culture given the next day, but this increased resistance induced by a single dose is transitory and has usually disappeared or at least is markedly diminished by the second or third day. However, the increased resistance induced by many successive doses is more lasting, manifesting itself for thirty days. The temperature curve of an animal previously treated with the soluble poison when inoculated with twice the fatal dose of the living culture is practically identical with that of an untreated animal when inoculated with less than a fatal amount of the living culture. This is shown in Chart IV.

CHART IV.



——— Curve of normal animal inoculated with non-fatal dose of living germ.
- - - - - Curve of immune animal inoculated with living colon bacill ♀ s.

In both these animals the minimum temperature is reached within from six to eight hours, after which it gradually ascends to the normal. This similarity suggests that we have here a condition similar to that generally spoken of as natural immunity, and this suggestion is supported by the fact that this increased resistance to living cultures of the colon bacillus may be secured by previous treatments of the animal with the poisonous groups split off from egg albumin and pepton as well as with that from the colon bacillus itself. It is quite certain that the increased resistance to the living culture secured by treatment with the soluble poison is not specific. This point is an interesting one, especially in view of the well-known fact, attested by several independent investigators, that increased resistance to certain pathogenic bacteria may be secured by previous intraabdominal injections of various proteid solutions.

Immunization with the non-poisonous residue of the colon bacillus.—I have stated that the portion of the colon cell left insoluble in alcohol after the cellular substance has been heated with a 2 per cent. solution of sodium hydroxid in absolute alcohol is non-poisonous. As much as 500 mg. of it may be injected into the abdominal cavity of a guinea-pig without visible effect. The temperature does not fall as it does after treatment with the poisonous portion, nor is there any measurable or constant rise. Still the non-poisonous residue must react with certain cells of the animal body; otherwise it would be difficult to explain the formation of an antibody. It seems from our work that the toxcity of a substance for the animal body as a whole depends upon the function of the cells in the animal affected by the introduced substance. The poisonous group of the colon bacillus seems to have a special affinity for the cells in the respiratory center of the animal, and consequently in sufficient dose it kills the animal speedily; while the non-poisonous part of the same bacterial cell acts upon other cells

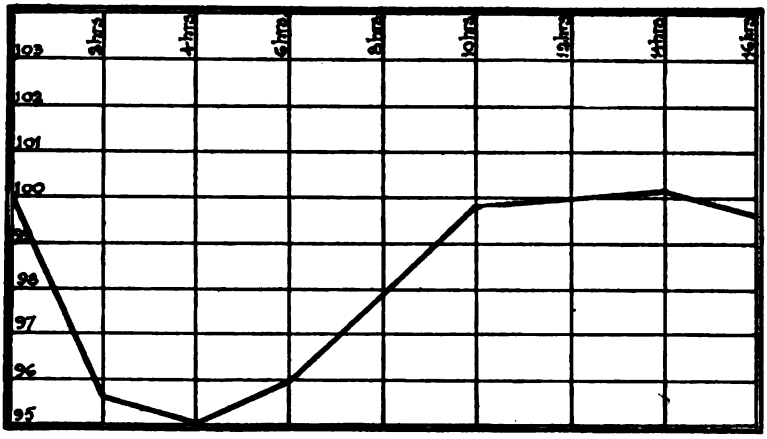
in the animal, and the function of these cells not being so immediately necessary for the continuance of the life of the animal no ill effects are directly observable. This is an important factor, and deserves farther study.

Guinea-pigs and rabbits treated with repeated doses of the non-poisonous part of the colon bacillus bear from six to eight times the minimum fatal amount of the living culture. The degree of immunity induced by these treatments depends not so much upon the total amount of the residue given as upon the number of injections and the length of time through which they are continued. There is, therefore, no object in using large doses of the residue in inducing immunity, because smaller doses given through a longer period induce a higher degree of immunity.

The clinical pictures obtained by immunizing animals to the poisonous and the non-poisonous portions of the colon bacillus differ in an interesting way. As has been stated, the symptoms induced in a pig by inoculation with twice the minimum fatal dose of the living culture after injections with the poisonous part are identical with those observed after inoculation of an untreated animal with less than a lethal dose of the culture. But animals immunized with the non-poisonous part and subsequently inoculated with twice the minimum fatal dose of the living culture are very ill within an hour, much sooner than the control, and after from six to eight hours, just when the control begins to manifest symptoms, the immunized animal begins to improve. All this is indicated in the temperature, as shown in the accompanying chart.

On this point one of my students, V. C. Vaughan, Jr., offers the following: "The difference between the behavior of animals treated with the toxic part and those which have been treated with the residue towards cultures of the living germ is easily explained, if we consider the fact that in the first case we are dealing with an animal which has acquired

CHART V.



a certain amount of tolerance for the intracellular poison of the colon bacillus as represented by the toxic part. In the case of animals treated with the residue, however, no tolerance for the poison contained within the colon bacillus has been developed. If now the process which takes place in both instances is a bacteriolytic one, it results that in the case of the animal immunized with the toxic group the effects of the poison contained within the bacterial cell and liberated upon its disintegration will not become manifest until a sufficient amount of poison has been set free to overcome the tolerance which the animal has attained during the process of immunization. In the case of the animal immunized with the residue there is no tolerance to be overcome other than that which is present in all animals, and the effects of the poison liberated through bacteriolysis become apparent sooner and to a more marked extent. Again, the fact that bacteriolysis may occur more rapidly in the case of residue pigs than in those immunized with the toxic group might explain in part the difference in behavior in the two cases. This is a point on which we are as yet unable to give any definite results.

"In order to study the differences in reaction to the living germ in animals treated with the toxic part and those immunized with the residue it is not only essential that they should receive the same amount of the same culture, but the dose given should not exceed twice that which would prove fatal for a control. When a larger amount of the living culture is given the differences are by no means so clearly defined, although even in this case the animal which has been treated with the residue shows symptoms of severity at a much earlier time. As can be seen from Chart V. the temperature of a residue pig which had been inoculated with twice the fatal dose of a living colon culture had begun to rise at an interval of six hours after injection. However, if an animal which has been rendered immune by treatment with the residue is inoculated with six to eight times the fatal dose of the living culture, we find that the temperature curve obtained is somewhat different in character. The temperature falls with the same initial rapidity, but instead of showing an early rise it continues for some time at a low point, and it is only at the end of from eight to ten hours that any appreciable rise is manifest. This, we think, is due to the fact that there has not been enough of the bacteriolytic substance directly available to destroy all the bacilli contained in the large amount of culture injected. The remainder of the germs are destroyed by the same factors which are operative in normal animals after the injection of a non-fatal dose of the living bacillus. As can be seen from Chart IV. it is only after an interval of six to eight hours that there is any appreciable fall in temperature in the case of a normal animal inoculated with a non-fatal dose of the living culture. This, we think, indicates that it is not until this time that any appreciable amount of poison is liberated by bacteriolysis, since, as we have seen in a previous paper, one of the first signs of the action of the intracellular poison is a fall in body temperature. In a pig

which has been immunized with the residue, and subseqently inoculated with a large amount of the living germ, we obtain evidence of hypothermia at a much earlier period, owing to the fact that bacteriolysis takes place very rapidly since the bacteriolytic substance is present in a form available for immediate use. If, however, the amount of this substance directly available is not sufficient to cause death and bacteriolysis of all germs present, those bacilli which remain are still capable of further reproduction. The same mechanism which causes destruction of the bacteria in normal animals, and which is probably connected with the phenomenon of phagocytosis, is, however, still operative in the immune animal. Thus we shall have two influences at work in the immune animal to cause bacteriolysis, one acting rapidly, and the other manifesting its action only after a considerable interval of time. We should, therefore, expect theoretically that we would find in the immunized animal a marked fall in temperature at an early time, due to the setting free of the poison from the bodies of the bacteria disintegrated by the directly available bacteriolytic substance followed by a secondary rise, and a succeeding fall due to the liberation of the poison by means of the factors present in the normal animal. However, this is not actually the case since the effect of the poison liberated at first has not worn off before the second period of bacteriolysis becomes well established. Consequently, the intermediate rise of temperature is absent."

Animals immunized against colon infection with the nonpoisonous residue manifest no increased tolerance to the soluble poison, but are quite as susceptible to this as untreated animals are. This indicates that this form of immunity is not due to the production of an antitoxin, but is due to increased capability of the body cells to destroy the bacteria. Whether the bacteria introduced into animals immunized with the residue are destroyed by some bacter-

iolytic substance increased in amount by the successive treatments with the residue and present in the fluids of the body or by increased phagocytic action, I am not yet in a position to determine with certainty, but I am inclined to the opinion that the latter is the important factor. It seems probable that in some way the phagocytic warfare against the invading bacterial cells is rendered more effective. On this point I hope to have in the near future more positive and convincing evidence.

The immunity induced by the non-poisonous residue differs in yet another important respect from that secured with the soluble poison. We have seen that the latter is not specific, but may be secured with the similar, if not identical, poisons obtained from egg-albumin and pepton. On the other hand, the immunity obtained with the non-poisonous residue is specific, and is secured only with the colon residue, and not with residues prepared from albumin and pepton. I conclude from this that the residue contains that group in the colon cell which is peculiar to that organism.

Part IV.—Summary and Conclusions.

I believe that this work which has been done by my students and myself on the chemistry of the colon bacillus and the action of the split products on animals, and which I have briefly outlined in the preceding pages, gives us a glimpse, as it were, of the physiology and pathology of the living cell. You will, therefore, please pardon me if I make an attempt to offer an explanation of some of these things. This will be difficult, somewhat tedious, and more or less unsatisfactory to both you and me. It will be difficult, in the first place, because the subject is a new one, and I shall be compelled to use terms which have been used with other meanings, and the speaker and the listener will be likely to understand the statements differently. How-

ever, I will do the best I can. As I have already indicated, I believe that life is molecular; *i. e.*, that the essential phenomena of cell life are due to intramolecular changes. This idea is not wholly original, for, as I have stated, the morphologists, such as Nussbaum, and the physiologists, such as Verworn, have expressed similar conceptions, although both have objected to saying outright that life is molecular, because the word molecule gives one the idea of a relatively stabile compound, while the living molecule is never, for any measurable period of time at least, in a condition of equilibrium. As I have stated, Verworn has proposed the expression "biogen molecule," but this can be interpreted only as a molecule that produces or induces life. I wish to acknowledge my debt to the very interesting monograph by Verworn on the Biogen Hypothesis, for from this I have obtained many of my ideas, and I intend this statement as a general acknowledgment of this fact, but I will not hold him responsible for my interpretations, with some of which he certainly will not agree.

I believe that the living cell is in its essential and active part composed of a chemical compound of very complex, but of definite, structure. This chemical compound contains a chemical nucleus, which must not be confounded with the morphological nucleus of the cell. The cell is composed of a chemical compound, not of a single molecule. There are many molecules in a cell, just as there are in a speck of salt, and there are as many chemical nuclei as there are molecules. I wish that I knew of some better term to use to designate the part of the cell molecule to which I refer, but I do not. By chemical nucleus I mean the centre of the chemism of the cell molecule; a centre of special chemical activity. This, as in all known proteid compounds, contains a benzol ring, and to it numerous side chains are attached. The presence of this benzol ring in the colon cell is shown by its response to the Millon test

and by the fact that we obtain aromatic split products, such as tyrosin and indol, by breaking up the cell with strong acids and alkalis. The tyrosin we have obtained in crystalline form, and have positively identified by determining its melting point and by distinctive tests, while we have produced the characteristic odor of indol by heating the cellular substance with strong alkali. We have also shown the presence of a carbohydrate group in the colon cell.

Verworn makes the following statement concerning the functional dissociation of the biogen molecule: "The nitrogen oxid (which exists as a side chain to the benzol ring), as happens in the manufacture of sulphuric acid, splits the molecular oxygen, which is brought to the living substance in the medium by which it is surrounded, and is converted into nitrogen dioxid. The nitrogen dioxid, when brought by the intramolecular movement of the atoms near to the aldehyde (carbohydrate) group (another side chain attached to the benzol ring) gives up an atom of its oxygen, and in this way carbonic acid is formed along with water, lactic acid and other simple non-nitrogenous bodies. The nitrogen, now reduced to nitrogen oxid, remains attached to the benzol ring, and again becomes oxidized to nitrogen dioxid." In this way a nitrogen side chain serves as a receptor and transmitter of the oxygen, and thus the traffic in energy within the living cell molecule goes on rythmically. It is not to be supposed that the nitrogen side chain, which serves as the receptor and transmitter of oxygen, consists of so simple a body as nitrogen or nitrogen oxid, but it is probably a highly complex nitrogenous body in which the location of the nitrogen is central, as suggested by Allen. Nor is it to be supposed that only oxygen is broken off from the cell pabulum, but substances containing this element. This is the way in which the living cell molecule keeps up its constant, rythmic traffic in energy, absorbing heat by assimilation and giving it off by dissoci-

ation. Each living molecule has not only one, but many, of these nitrogenous side chains that act as receptors, and not only one, but many, carbohydrate side chains that furnish carbon and hydrogen for oxidation. Moreover, the metabolism within the molecule is not confined to the absorption of oxygen and the casting out of non-nitrogenous products of combustion. The whole molecule is labile, and there is probably in every living cell molecule a nitrogenous as well as a non-nitrogenous metabolism. The nitrogen absorbed with the oxygen is utilized in replacing the waste in this element, and the carbon brought into the molecule at the same time is in part detached by the free valences in the carbohydrate side chains and used to repair the loss in this part of the molecular structure. In the cell molecule it is probable that the nitrogenous metabolism takes place much more slowly than the carbon and hydrogen metabolism, and in both it goes on rythmically, and the tempo with which it proceeds depends upon the swing of the atomic groups that constitute the molecule, and this rate can be changed, hastened or retarded, by alterations, either physical or chemical, in the medium in which the cell lives. Under certain conditions all metabolism in certain cell molecules may be indefinitely arrested without disruption of its structure. The cell molecule is then not actively alive, but is possessed of latent life. This is true of spores, seeds and ova. In a seed, as I have already stated, the germ of life; *i. e.*, the molecular structure, is present, but there is no activity, probably because there is nothing in the seed to which the molecular side chains or receptors can attach themselves, but when the seed is placed under suitable conditions the ferment present in the pabulum surrounding the germ breaks up the complex bodies into simpler bodies, which react with the receptors, and active life is awakened. Even when the cell molecule is in active life its food is prepared for it by ferments, and there are many

who now believe that every kind of cell has its own special ferment. Be this as it may, it is certainly true that before a cell molecule can use outside matter in its reconstruction that outside matter, or pabulum, must be split up into relatively simple bodies. This is in part accomplished in animals by digestion in the alimentary canal; and in each organ this preparation of the food material is more specifically carried out by ferments supplied by the cells of each organ. It is most probable, indeed it is quite certain, that these ferments have their origin in the nitrogenous metabolism of the cell molecule. Certain side chains are detached from the living molecule, and in solution pass into the surrounding medium, splitting up complex bodies and fitting them for the receptors of the molecule.

I might enter into greater detail concerning the chemistry of the cell and my conception of the way in which it functions, but I am endeavoring to make this statement as brief as possible, and I think that anyone who has followed me so far can get my point of view, which, I take it, is not essentially different from the teachings of Ehrlich and Verworn.

I will now attempt to interpret the results of the investigations on the chemistry of the bacterial cell, and in order to avoid exhausting your patience I will condense as follows:

(1) The cellular substance of the colon bacillus, after being freed from all matter held by it and in it mechanically, consists of a complex chemical compound composed of chemical nuclei with numerous attached groups or side chains.

(2) The chemical nucleus, or centre, of chemism is contained in our "crude soluble poison." When the cellular molecule is split up by means of an alcoholic solution of sodium hydroxid, the molecular nucleus is found in that fraction which is soluble in alcohol.

(3) Since we have obtained similar "soluble poisons" from all the bacteria with which we have worked, also from egg-albumin and pepton, I conclude that the chemical nucleus in all these bodies is similar, but not identical. The presence of the benzol ring in the soluble poison is shown by its prompt response to the Millon test. However, the chemical nucleus consists of more than the benzol ring; it contains the benzol ring with the receptor nitrogenous side chains, and our soluble poison also contains the benzol ring with the nitrogenous receptors.

(4) The chemical nucleus has great affinity for other substances containing groups from which it can detach side chains, and for this reason is an active cell poison.

(5) The modus operandi of the chemical nucleus as a poison is explainable by its detaching from certain cell molecules of the animal body side chains, upon which the functioning of the animal cell depends.

(6) The intensity of the poisonous action of the chemical nucleus depends in the first place upon the intensity of its chemism, and in the second place upon the number and condition of its receptors. Our method of detaching the chemical nucleus and rendering it soluble is crude compared with the process as it occurs in the animal body. Five milligrams of colon germ substance thrown into the abdominal cavity of a guinea-pig kills, while the smallest fatal dose of soluble poison that we have obtained is eight mg., representing about twenty-five mg. of the germ substance. In our crude attempt to isolate the chemical nucleus we have probably destroyed, or at least inactivated, many of its receptors. Our soluble chemical nucleus acts much more promptly than the cellular substance, because we have broken up the cell molecule and set the nucleus free, which process in the animal body goes on slowly, but with less loss. The receptors of the chemical nucleus of the colon bacillus are acid in character and can be neutralized with

sodium bicarbonate, but when the sodium compound is introduced into an animal the alkali is replaced by atomic groups, for which the receptors have greater affinity, and these groups being taken from body cell molecules, the neutralized chemical nucleus is a poison with the same action as the free nucleus, but less prompt.

(7) The chemical nucleus of all the bacterial cells with which we have worked, as well as that of egg-albumin and pepton, appears to have a special affinity for certain side chains in the cell molecules of the respiratory centre of the animal. It tears off these side chains from the cell molecules of the respiratory centre, thus interrupting the functions of these cells and causing the death of the animal.

(8) It is probable that the chemical nucleus of every foreign cell, and of every foreign proteid as well, when brought into the circulation in a free state, is more or less of a poison to the respiratory centre.

(9) Whether a given proteid will prove poisonous to an animal or not, when introduced into the blood directly by intravenous injection or indirectly by subcutaneous or intra-abdominal injection, depends upon whether or not its chemical nucleus is set free intact and upon the rapidity with which it is set free.

(10) The chemical nucleus of a cell or a proteid does not absolutely destroy the cells of the animal body. It tears off their side chains, and impairs or interrupts their functions. If these side chains be detached from the animal cell molecule slowly and in only a few cells at a time, reconstruction lags behind destruction but little and the life of the animal is not endangered, but when these side chains are detached, as it were, in mass and from a large portion of the cell molecules of a given organ, then the function of that organ is perceptibly impaired, and it may be completely interrupted, and if this happens in an organ whose continued functioning is essential to the life of the animal, death results immediately.

(11) The chemical nucleus of the colon bacillus, at least, when administered by the alimentary canal, or when introduced into the abdominal cavity enclosed in a collodion sac, diffuses so slowly that it does not visibly affect the animal. It is also, in part at least, destroyed in the intestine, when taken by mouth. Furthermore, it is possible that the chemical nucleus even of the proteid foods largely passes through the alimentary canal, and is not absorbed at all.

(12) When the chemical nucleus is broken up its constituents are not poisonous, and inasmuch as it is a labile body its conversion into a harmless substance is easy under favorable conditions.

(13) The chemical compound of which the essential part of the colon bacillus is composed is not a poison, or at least not an active poison, until it is disintegrated by the aggressive action of the cell molecules of the animal body. The hemorrhagic peritonitis observed after death from intra-abdominal injection of the colon bacillus is not induced by, nor is it connected directly with, the growth and multiplication of the bacillus, for it is just as marked after the injection of the dead bacterial substance as it is after inoculation with a living culture. The hemorrhagic peritonitis results from the chemical reaction between the constituents of the bacterial cell and certain constituents of the body cells.

(14) That the soluble poison obtained by splitting up the colon cell is the poison that kills when an animal is inoculated with a living culture, is indicated by the following:

(*a*) The effect on the temperature. This is the same whether the animal be treated with a living culture, the dead germ substance or the soluble poison, except in the interval of time that elapses between the introduction of the foreign substance and the beginning of the fall, and when the soluble poison is combined with an alkali this period is lengthened.

(*b*) If with either of these the dose employed is less than a lethal one, the first trustworthy evidence of recovery is a rise in temperature.

(*c*) When a lethal dose is employed, the mode of death is the same; *i. e.*, it is due to failure of respiration.

(*d*) When the soluble poison is combined with alkali and time enough given for the saturation of the receptors with the alkali, the post mortem findings are the same.

(15) The production of increased tolerance, and the formation of an antibody, should further experimentation show that such a body can be produced, by successive treatments of animals with the soluble toxin, can be explained by Ehrlich's theory, or by a modification of this theory, which I have elsewhere discussed.

(16) Concerning the immunity secured against the living culture by successive treatments with the non-poisonous residue of the colon bacillus, I wish to say a few additional words. This I regard as the most interesting part of our work. It should be remembered that in these animals there is no increased tolerance to the soluble poison, and we must conclude that this form of immunity is due to bacteriolytic or phagocytic action. I have spoken quite at length concerning the chemistry of the bacterial cell, and I assume that the cells of the animal body are made up in a similar manner of living molecules, each of which contains a chemical nucleus with numerous side chains attached. These animal cell molecules split off from the bacterial cell molecules side chains upon which the functioning of the bacterial cell depends, and thus deprive them of life. Now by feeding the body cells with the detached side chains of the bacterial cell molecules the former are sensitized, as it were, to this kind of food, and acquire greater avidity for it with each successive administration. The chemism of the animal cell for these special side chains is intensified, and when the living culture is introduced the advantage is with

the body cell the lability and chemical structure of which have been adjusted to the absorption of the side chains of the bacterial cell molecule. This is probably due to some alteration in the chemical structure of the receptor side chains of the animal cell molecule. We recognized early in our work that if we could secure a perfect condition of this nature, enough poison should be set free in a short enough time, when a large amount of dead germ substance was introduced into the abdominal cavity, to kill the animal in the same time that it takes the free soluble poison to kill, and we made many attempts to reach this result. On this point V. C. Vaughan, Jr., in a paper published in 1905, wrote as follows: "Although we have as yet been unable to actually cause death in an immune guinea-pig at an early period, the animals are in every instance very ill within thirty minutes after the injection of the dead culture. In fact, several of them have shown signs of the commencement of the convulsive stage, as evidenced by slight convulsive movements of the head separated by considerable intervals of time. We have been unable to secure a fatal result in these animals up to the present time, simply because we have worked with pigs which did not possess a sufficient amount of bacteriolytic substance directly available to cause disintegration of enough bacilli to liberate a fatal amount of poison at one time. It is worthy of note that this behavior of animals immunized with the residue towards the dead bacterial substance furnishes additional proof of the fact that the poison of the colon bacillus is an intracellular one. If the poison existed free in the culture medium, we should expect that the control would show evidence of its action at as early a period as does the treated animal. However, as has been stated, this is not the case. The fact that the treated animal shows symptoms of poisoning to a much greater degree and at an earlier time than does the control, can be explained only on the ground that

the poison with which we are dealing is an intracellular one and is set free only after the disintegration of the bacillus by bacteriolysis."

It seems quite evident that the animal cell molecule does not absorb or destroy the chemical nucleus of the bacterial cell molecule, but when the other side chains are torn from the bacterial cell the chemical nucleus goes into solution. It should be understood that for the body cell molecule to break up the bacterial cell molecule it is not necessary for the two to come in contact in cellular form. Indeed, bacterial cells may be dissolved in body fluids in which there are no animal cells, but I imagine that this is due to ferments that have been formed in the body cells. As we have seen, the more complex bodies in the pabulum are broken into simpler bodies by soluble ferments, and I do not suppose that there is any intermolecular reaction between cells except of substances in solution.

PART V.—BIBLIOGRAPHY.

The following are the more important papers from the Hygienic Laboratory of the University of Michigan bearing on the subject discussed in the preceding pages:—

(1) Ein in Eiscreme und Käse gefundener giftproducirender Bacillus. By V. C. Vaughan and G. D. Perkins. Archiv. f. Hygiene. 1896. 38, 308.

(2) Bacteriologic Poisons in Milk. By V. C. Vaughan and J. P. McClymonds. Jacobi Festschrift, p. 108.

(3) The Bacterial Toxins. By V. C. Vaughan. Transactions of Ass. of Amer. Physicians. 1901.

(4) The Toxin of the Colon Bacillus. By V. C. Vaughan. The Journal A. M. A., Feb. 23, 1901; Amer. Med., May, 1901.

(5) The Toxcity of the Dry Sterile Cells of Certain Non-Pathogenic Bacteria. By A. J. Detwiler. Trans. Assn. Amer. Phy. 1902.

(6) The Chemistry of Sarcina Lutea. By May Wheeler. Ibid.

(7) The Chemistry of Bacillus Coli Communis. By Mary F. Leach. Ibid.

(8) The Toxcity of the Cellular Substance of the Colon Bacillus. By C. E. Marshall and L. M. Gelston. Ibid.

(9) The Intracellular Toxin of the Diphtheria Bacillus. By L. M. Gelston. Ibid.

(10) The Anthrax Toxin. By J. W. Vaughan. Ibid.

(11) The Action of Mineral Acid on the Cellular Substance of Bacillus Typhosus. By May Wheeler. Journal A. M. A., 1904. 13, 1000.

(12) The Chemistry of Bacillus Coli Communis. By Mary F. Leach. Ibid, p. 1003.

(13) The Intracellular Toxin of Bacillus Pyocyaneus. By D. McIntyre. Ibid, p. 1073.

(14) The Extraction of a Toxin from Liver Cells. By J. F. Munson and F. R. Spencer. Ibid, p. 1075.

(15) Further Studies on the Intracellular Bacterial Toxins. By V. C. Vaughan. Journal A. M. A. 1905.

(16) The Extraction of the Intracellular Toxin of the Colon Bacillus. By May Wheeler. Ibid.

(17) The Action of the Intracellular Poison of the Colon Bacillus. By V. C. Vaughan, Jr. Ibid.

(18) The Production of Active Immunity with the Split Products of the Colon Bacillus. By V. C. Vaughan, Jr. Journal of Med. Research. 1905. 14, 67.

(19) On the Chemistry of Bacillus Coli Communis. By Mary F. Leach. Journal of Biological Chemistry. No. 4. 1906.

ARTICLE XVI.

REMOVAL OF AN ENORMOUS OVARIAN TUMOR.

By WALTER A. SMITH, M.D.
OF SPRINGFIELD.

READ JUNE 13, 1906.

REMOVAL OF AN ENORMOUS OVARIAN TUMOR.

My only excuse for presenting this case is the large size of the tumor. The fact that it was a simple cyst, with a very small pedicle, serves to show still more emphatically the harm that can be, and is still being done, by the quacks that are allowed to practise in our community.

Patient, Mrs. A. H., age 42. History of illness dates back two or three years, since which time she first noticed she was increasing in size. She had been under treatment by a quack who applied electricity, static and other varieties, for one to two years. Following this she consulted an osteopath who refused to treat her. Later for a few months she tried mental science, though I understand they advised a surgeon; following which she consulted a Christian Science healer who I understand assured her that all that was needed for a cure was faith in her nefarious cult.

I first saw her May 15th. She presented the appearance seen in the accompanying photograph. I at once made a diagnosis of ovarian cyst and she was removed to the Springfield hospital the next morning. The patient's measurements were, height 5 feet 4 inches, weight 375 pounds, waist measure 204 c.m., ensiform cartilage, to pubes 94 c.m. Examination of urine, no albumen or sugar, specific gravity 1015. 3 P.M., patient's pulse 120. I introduced a trochar and drew off 14 gallons of fluid that weighed 126 pounds. Time of operation about two hours. Pulse

rate at close of operation, 100. Present at the operation, Drs. C. P. Hooker, R. H. Seelye, J. B. Comins, I. R. Calkins, E. C. Collins and Dr. Eaton.

Patient passed a fairly comfortable night, morning pulse 100. At 8.30 May 17th, the usual abdominal incision was made for the removal of the cyst.

I found the cyst wall adherent over the entire front of the abdomen, covering a space of about three square feet, extending to the diaphragm, and involving a portion of it. I think the adhesions were caused in great measure by the electric treatment above referred to. Underneath the liver was a mass the size of a child's head consisting of several small cysts. The pedicle was about ½ inch in diameter and I was only obliged to ligate one band of adhesion.

The adhesions seemed to furnish most of the blood supply, and bled very freely for a moment only after being separated. Large quantities of normal salt solution were injected into the tissues during the operation and also used for flushing the abdominal cavity.

As we removed the patient from the operating table, we placed the stretcher on the scales and she weighed 183 pounds; deducting this from 375 pounds, her weight the evening before, gives 192 pounds, the weight of the cyst and contents.

I wish to express my obligations to Drs. C. P. Hooker, J. B. Comins, I. R. Calkins and Dr. Eaton, house surgeon, who rendered me all the assistance possible. Those present at the operation were, Drs. C. P. Hooker, R. H. Seelye, R. B. Ober, and others.

Patient died 24 hours later from general exhaustion and shock following the operation.

ARTICLES XVII. AND XVIII.

DISEASES OF THE BILIARY PASSAGES, INCLUDING THE LIVER, THE GALL BLADDER AND THE PANCREAS.

BY

ELBRIDGE G. CUTLER, M.D.

AND

MAURICE H. RICHARDSON, M.D.

OF BOSTON.

READ JUNE 13, 1906.

ARTICLE XVII.

DISEASES OF THE BILIARY PASSAGES AND PANCREAS.

By ELBRIDGE G. CUTLER, M.D.
OF BOSTON.

For convenience of consideration we may group the most important diseases of the biliary passages under four heads.

1. Calculi or gall stones in the gall bladder and gall ducts and their effects.

2. Inflammation of the gall bladder and gall ducts and their effects.

3. New growths of the gall bladder and gall ducts and their effects.

4. The various combinations of these three groups.

These diseases are not equally frequent. Riedel and Kehr say that culculi of the biliary passages are found in ten per cent. of all adult autopsies. Robson states that post mortem records on persons of all ages and both sexes prove gall stones to be present in from five to ten per cent. of all Europeans; in Strasburg the record being twelve per cent., in Kiel five per cent., and iu Manchester seven and four-tenths per cent. Mosher found in the first sixteen hundred and fifty-five autopsies of the Johns Hopkins Hospital there were one hundred and fifteen cases of gall stones or six and nine-tenths per cent. Rolleston found them in five and seven-tenths per cent. in the post mortem records

of the St. George's Hospital in London. Brewer found them twelve times in one hundred subjects examined in the dissecting room of the College of Physicians and Surgeons, New York. Wright in his first fourteen hundred autopsies, at the Massachusetts General Hospital, found there were 120 cases of disease of the biliary passages and pancreas. Of these 35 were stone, 9 thickened bile, 6 cholecystitis, 4 adhesions of gall bladder to intestine, 3 small and contracted gall bladder, and 1 each hemorrhagic pancreatitis, stricture of ducts and cancer of the common duct. Among these cases were 3 of cancer of the gall bladder with gall stones, 3 of cancer of the pancreas without stone, 1 with cancer of the pancreas and secondary cancer of the common duct, 1 of lymphosarcoma of pancreas, gall bladder and liver with stone. In view of the greater frequency of gall stones I shall devote myself, in the short time at my disposal, more particularly to that form of disease.

In by far the greater number of gall stone cases there are no symptoms. Kehr says ninety-five per cent. are without them, and one or more stones may remain indefinitely without symptoms in a healthy gall bladder with pervious gall ducts. In the remaining five per cent. symptoms result which vary from the very slightest evidences to fatal consequences.

The chief symptoms produced by these above groups of disease are pain, local tenderness, chills and fever, vomiting, sweating, alteration of the rate of the pulse, jaundice, tumor of the gall bladder, changes in the urine, blood and stools.

Pain.—The pain is acute, paroxysmal, in the right upper quadrant, though it may be in the left according to Naunyn, occurs more often when the stomach is empty, frequently comes on at night, in most cases is referred to the stomach region and begins in the epigastrium and not in the region of the gall bladder (Ewald). Sometimes it is

present all over the abdomen, it extends often to the right shoulder and back both sides of the vertebræ, it is frequently relieved by eating, and sometimes by the escape of gas from the intestine. Its duration, frequency and severity are very irregular. Kehr and many authorities claim the pain to be dependent on inflammatory changes. Riedel says it is due to the advance of a stone in scarcely half the cases, but is caused by inflammation. Murphy, Furguson and others say that it is due to the effects of gall stone or to distension of the gall bladder or ducts independent of infection. Ewald says colic is caused by contractions. Characteristic is the sudden onset, its colicky nature, its sense of boring, tearing, through. There is nothing peculiar to the pain *per se* which enables us to distinguish between inflammation, stone and cancer, though its accompanying symptoms will often enable us to make a diagnosis.

Tenderness.—The next most characteristic symptom is local tenderness in the part involved in the disease. Its greatest intensity is just underneath the free border of the ribs at the junction of the ninth rib and its cartilage. It is best elicited, according to Moynihan, by bimanual examination, the left hand pressing firmly up from behind the lower two or three ribs and the fingers of the right hand hooking under the costal margin while the patient draws a long breath. Robsen's point is above and to the right of the umbilicus at the junction of the upper two-thirds with the lower one-third of a line drawn from the ninth rib to the umbilicus. He says it is the site of reflected pain passing along the splancnic to the eighth and ninth dorsal nerves and thence reflected to the surface of the termination of these nerves. Tenderness is a sign of considerable importance. The tenderness is generally nearer the median line and slightly lower when the common duct is the seat of trouble.

Fever and Chills.—With the attack of colic, when due to stone, a severe rigor not infrequently occurs and may be repeated several times. It is accompanied by a rapid rise of temperature to 102° F., 103° F. or more and with the subsidence of the pain it as rapidly disappears. Murphy has called attention to the fact that the temperature is a series of angles not curves. When inflammation of the gall bladder or ducts or their sequences have occurred fever is constant with exacerbations.

Vomiting.—Vomiting is of very frequent occurrence in an acute attack and follows the pain. It may be the most troublesome of the symptoms. It is reflex and may be independent of nausea. Its severity may be so great that a gallstone may be dislodged from the straining with temporary subsidence of the attack.

Sweating.—Sweating may occur during the acute attacks of gallstone colic or it may be the result of septic infection.

Pulse.—The pulse is quickened and its tension increased during an acute attack of gallstone colic; while in inflammation of the gall bladder or ducts it is simply accelerated and the tension may be diminished.

Jaundice.—Jaundice is one of the least constant of the symptoms. According to Brewer it is absent in from 80 per cent. to 90 per cent. of all operative cases. It may be caused by stone in the hepatic duct, or in the common duct, by inflammation of the hepatic or common duct, as a late symptom in cancer of the gall bladder, tumors of the hepatic duct, tumors of the common duct or neighboring viscera, by localized peritonitis distorting or constricting the common or hepatic ducts. It may be the result of functional disturbance (Ewald). Prolonged, persistent, deepening jaundice with progressive loss of weight and tumor of gall-bladder is strongly suggestive of cancer, though it has been observed in impacted stone in the common duct.

Tumor of gall bladder.—Tumor may be found in inflammation of the gall bladder, in concretions in the same, and in obstruction of the cystic or common duct. It projects below the edge of the ribs and may be felt as a mass of almost any size, pyriform, moving with respiration, may or may not be tender, and may or may not be hard. Often its lower end is parabola shaped and gradually disappears toward the liver.

Urine.—The urine may or may not contain bile either temporarily or constantly. It frequently contains albumin as well; albumin is always found immediately after an attack of biliary colic due to stone and may rapidly disappear.

Blood.—Leucocytosis may be found in suppuration in all infections save those of typhoid fever (present in 50 per cent. Musser).

Stools.—Examination of the stools determines the presence or absence of bile and degree of jaundice. Gall stones may be found a few days after an attack of colic, even as late as the fourteenth day (Robson).

Disease of the biliary passages is to be differentiated from gastric ulcer, appendicitis, renal colic, pancreatitis, angina pectoris, portal phlebitis, subphrenic abscess; gastric crises of tabes.

In *gastric ulcer* there is permanent hyperacidity and hyperchlorrhydria. In gall bladder colic chemical examination of the gastric contents during the interval shows no change from the normal. During or immediately after the attack there may be increase of the hydrochloric acid in the stomach, which disappears with the subsidence of the attack. In ulcer, injestion of food brings on the pain regularly; whereas that of gall bladder colic is irregular, appears at varying intervals and frequently at night. Cholecystitis is frequently mistaken for perforation of peptic ulcer.

Appendicitis is sometimes difficult to differentiate, epecial-

ly when the appendix occupies an abnormal position with its tip directed upward and perhaps near the liver. Inflation of the colon may be of service by displacing the painful point. Leucocytosis points here to appendicitis as empyema of the gall bladder has no leucocytosis (Ewald).

In *renal colic* the pain originates in the flank, extends down along the ureter and ends in the testis or ovary, and there is frequently hæmaturia and vesical irritation.

Pancreatitis.—It may be impossible to distinguish between acute pancreatitis and acute cholecystitis or phlegmonous cholecystitis. As a rule, the clear description of Fitz will enable us to keep in the right direction.

Angina pectoris could only be confounded with biliary disease in irregular cases.

Portal pylephlebitis is distinguished by its antecedent history, its enlarged and painful liver and its pyæmic symptoms.

Subdiaphragmatic abscess, the previous history, the physical signs (abdominal as well as thoracic), and exploratory puncture would serve to differentiate.

Gastric crises of tabes.—The other accompanying nervous symptoms would stamp the disease.

This short resumé illustrates the occasional difficulty of diagnosis, and, as said before, an absolute diagnosis may sometimes be impossible.

The diagnosis having been made, however, the indications from a medical point of view are to alleviate symptoms, to promote a free passage of bile to the intestine and to prevent stagnation of bile. The first of these indications is met by the well-known means and may be successful. They are for the pain of gall stones the subcutaneous injection of 1-4 grain of morphia or better, morphia gr. 1-4, atropia gr. 1-150, nitroglycerin gr. 1-100 repeated without the atropia in 15 to 20 minutes if relief is not obtained in the first instance. Hot bath and hot fomentations to the

hepatic region. Inhalation of ether or chloroform till the morphia has begun to act. Rest in bed and local applications till the tenderness subsides.

The promotion of the free passage of bile to the intestine and the prevention of concentration and stagnation in the gall bladder is thought to be favored by the giving of some of the soda salts as the sulphate or phosphate in doses of from 1 to 2 drachms daily. Oleate of soda in pill form has received the indorsement of Robson in cases that declined or were unfit for operation. The attempts to dissolve the gallstones in situ have not thus far been successful. Something may be accomplished in the way of prophylaxis by regulation of diet, prescribing regular exercise, the use of saline purgatives and care of the general health, but after all the case is almost always sooner or later sure to come into the hands of the surgeon, and it is far wiser to call him in early as soon as the diagnosis is established and together to determine on the future course rather than to wait till some serious complication has arisen and the surgeon must work at a serious disadvantage.

This bibliography simply indicates the writers referred to in the text, and by no means pretends to be either complete or to contain the names of all those consulted by the compiler.

BIBLIOGRAPHY.

RIEDAL: Erfahrungen über die Gallensteinkrankheit mit und ohne Icterus. Berlin, 1892. Berliner Med. Wochen. Bd. xxxviii, s. 1, 1901.

NAUNYN, B.: Klinik der Cholelithiasis. Leipzig, 1892.

BROCKBANK, E. M.: On Gall Stones. London, 1896. Edinburg Med. Jour., vol. lii, p. 51. 1898.

ROBSON: Diseases of the Gall Bladder and Biliary Ducts, including Gall Stones. 1904.

MUSSER, J. H.: Boston Med. and Surg. Journal, vol. cxxi.
Trans. Congress American Physicians and Surgeons. 1903.

MOYNIHAN: Gall Stones and their Surgical Treatment. W. B. Saunders & Co. 1904.

MOSHER: Johns Hopkins Bulletin. Aug., 1901.

ROLLESTON, H. D.: Diseases of the Liver, Gall Bladder and Bile Ducts. W. B. Saunders & Co. 1905.

BREWER: Trans. of the Congress of American Physicians and Surgeons. 1903.

WRIGHT, J. H.: Autopsy Records, Massachusetts General Hospital. Oct. 19, 1896, to April 21, 1905.

KEHR: Gall Stone Disease. American Translation. 1901.
Trans. Congress American Physicians and Surgeons, vol. vi. 1903.

EWALD: Transactions Congress of American Physicians and Surgeons. 1903.

MURPHY, J. B.: The Diagnosis of Gall Stones. Medical News, May 2, 1903, p. 828.

MAYO, W. J. and C. H.: Trans. Congress of American Physicians and Surgeons, vol. vi. 1903.
Northwest Lancet, Minneapolis. 1905. xxv, p. 10.
St. Paul Med. Journal. 1905. vii, 79–85.
American Journal Medical Sciences. 1905. ns., cxxix, 375–380.

KEMPERER, G.: Therapie der Gegenwart, Berlin. 1905. xlvi, 76–80.

HERTER: Transactions Congress American Physicians and Surgeons, vol. vi. 1903.

ARTICLE XVIII.

DISEASES OF THE BILE PASSAGES, INCLUDING THE LIVER, GALL-BLADDER, AND PANCREAS.

By MAURICE H. RICHARDSON, M.D.
OF BOSTON.

In numerous manual and digital explorations of the right upper quadrant, in the course of abdominal operations undertaken for other purposes, I have found unsuspected lesions, chief among which have been diseases of the biliary tract dependent upon gallstones. In one or two cases I have found cancer of the pylorus, and in one or two serious disease of the liver itself. The number of pathological lesions detected in patients who have never complained of this region, has been rather surprising. The operations in which these lesions were discovered were such as required incision large enough to admit the hand and arm. I therefore have been able to make careful digital examinations. It has so often been said that gallstones may exist for a long time without causing any symptoms that I have been interested in investigating the previous histories in the light of the digital demonstrations. Many interesting facts have thus been brought to light.

It has been frequently said that this, that, or the other subject has been exhausted, and that nothing new can be

written upon it,—that, in fact, the matter of gallstones has been threshed out until nothing but a rehash of old facts can be expected. Such remarks—to those of us who are actively engaged in gallbladder work, who see daily our shortcomings in everything pertaining to it, who find something new in every case, something which we feel we ought to bring to the attention of others—far from discouraging discussions like these, cannot but stimulate them, if for no other reason, to prove to the over-confident that there is much more ignorance than knowledge in our daily routine contact with diseases of the liver, biliary tract, and pancreas.

And yet those who think they know a little, at least, of these matters are surprised every day to realize how small that knowledge is. I could, I am sure, give details in the matter of gallstones alone which would show that this subject is far from being exhausted. Take, for example, the diagnosis of gallstones,—how often is a gallstone as cause for acute intestinal obstruction even suspected? In a recent case of this condition, operated upon by Dr. Clara Alexander, I had made, after careful study, a diagnosis of cancerous obstruction in the sigmoid flexure, and had advised exploration, under a prognosis so unfavorable that many a family would have rejected operative treatment. On the other hand, how often do we find under a diagnosis of gallstones, some other and inoperable lesion? Having found in the biliary passages an adequate cause for symptoms, who would go farther and examine remote regions of the abdomen, especially when such exploration necessitates an enlargement of the incision?

If these questions are not hard to answer, what will be said of certain others: What does a distended gallbladder without symptoms mean? Can one fail to detect gallstones in a normal gallbladder that he holds between his fingers? What is the real connection between gallstones and pan-

creatic lesions? In doubtful cases, would you incise the pancreas in the hope of finding a gallstone instead of a cancerous nodule? What is the nidus of gallstones? Do they ever form in the liver; and, if so, how far may the surgeon reasonably go in search for them? By what anatomical appearances can the surgeon recognize the difference between a chronic inflammation and a malignant infiltration of the gallbladder? In a given case, is the origin of extensive and disabling adhesions about the pylorus the result of gastric ulcer or chronic cholecystitis? Is there such a thing as suppression of bile; if so, what is its cause and what are its effects? Who knows, when his hand and eye are on the liver, whether it is enlarged from obstructive biliary engorgement owing to gallstones, from hypertrophic cirrhosis, from syphilis, or from cancer? Who, indeed, even if he is a trained pathologist, knows much about the anatomical appearances during life in any of the diseases included in this discussion?

What, in general, is the connection between symptoms and anatomical changes? What is the connection between marked anatomical changes and questionable histories? What causes pain in gallstones, and why is it so often absent, even when the pathological changes are most extensive?

What is the cause of persistent and uncontrollable capillary hæmorrhage after operations upon the biliary tract? "Jaundice, of course," will be the reply. What, then, is the cause of such hæmorrhage after operation for biliary fistula of long standing, when there is no bile either in the blood or in the intestines? In a word, what do we really know about the early symptoms of gallstones, and how confidently may we expect to recognize them? When we have recognized them, how great certainty of diagnosis shall be required before we explore, and how much trouble must they cause? To all these questions the really trut hfu

answer must be an admission of ignorance more or less complete. Not wishing to weary you with these unsolved problems, the very mention of which would take all the time at my disposal, I would say to those—if any there are—who look upon these matters as settled, that I for one feel that they are far from a correct solution; and that it is only by renewed study, by many discussions, especially those based upon the disagreeable facts of failure, that we can hope to gain the knowledge which will give us that confidence in diagnosis, indications for treatment, and operative technique which we have learned to feel in that vastly more simple field—the right iliac fossa.

The scope of this paper does not permit my going into details of cases upon which questions like these are based. Every suggestion of ignorance which I have mentioned is based upon actual experience in the operating-room. One or two cases, however, will suffice to illustrate the origin of my questions.

In a stout woman of forty-four (O. R. 36—27), after removal of a large fibroid, I found in the gallbladder two stones which together were about the size of a pullet's egg. The gallbladder was apparently little, if at all, changed, either to touch or sight. It was more or less contracted upon these stones. After the convalescence of this patient I inquired into the history with especial reference to them. The patient said: "I have never had any pain or other symptoms connected with the liver; but of course I have always been subject to these severe bilious attacks, and I have to be very careful what I eat!" It was perfectly clear that the "bilious attacks" were painful contractions of the gallbladder upon these stones. They still remain, however, and will be removed as soon as the patient makes up her mind to undergo the operation. The chief danger in this case while the stones remain is cholecystitis and perforation of the gallbladder.

In the case of a second patient, now convalescing from an exploration, the symptoms of gallstones have been of but few weeks' duration (vol. lxii., p. 67). This was astonishing to me, for the gallbladder was packed from the fundus to the common duct with stones of great size, and the patient was deeply jaundiced. She had, besides the gallstones, hopeless cancer of the liver and of the parts about the foramen of Winslow. She repeatedly said that she had never had any symptoms whatever of gallstones; but, on persistent questioning after the operation, she finally recalled that twenty years before she used to have "severe bilious attacks." The doctor said that the attacks were "part bilious and part dyspepsia." The patient was a woman of about sixty: the stones were of ancient origin—they were black and hard, as old gallstones usually are. These stones undoubtedly gave rise to symptoms which were treated and described twenty years before as "partly bilious and partly dyspeptic."

I mention this case to show also one of the great dangers of gallstones of long standing; for it might be said that, in spite of her stones, the patient had had twenty years of life during which the only symptoms were those of dyspepsia and biliousness; whereas she might have suffered much more from the effects of perhaps a poorly performed or inadequate operation—or might even have lost her life. The irritation of the stones have, however, produced in her what they have produced in many others—hopeless malignant disease.

In a woman of fifty-seven (vol. lviii, p. 161), during an operation for a large ovarian cyst, I found the gallbladder so tense that it was impossible to produce any impression upon it by the fingers. It felt as if it were the seat of chronic cholecystitis, with walls thickened and contracted upon its contents, but with no stone perceptible in the cystic duct. Was this a diseased gallbladder? Is the normal

gallbladder ever so firmly contracted as to permit no impression of the fingers? There might well have been, in the small cylindrical viscus, gallstones which I could not feel.

A young woman (vol. lxii., p. 57) with symptoms suggesting now gastric ulcer with strictured pylorus, now gallstones, was explored on May 7, 1906. The stomach, pylorus, duodenum, hepatic flexure of colon, gallbladder, and right kidney were carefully examined. Nothing was found except a gallbladder, overdistended, with a small, soft stone in the beginning of the cystic duct. The gallbladder was drained for a fortnight. During convalescence the epigastric symptoms continued, with vomiting. The patient went home on June 2d. Still the symptoms continued. The attending physician, a man of large experience and marked common sense, insisted on a gastric cause. He did not, however, see the operation. The patient now vomits continuously, is emaciated, constipated, and suffering. Gastric ulcer, adhesions, twist of transverse colon, contracted pylorus, and neurosis are given by different observers as causes for symptoms. Is there nothing to learn in a case like this when no two observers agree either as to diagnosis or treatment? *

* July 5th.—In this case Dr. Tilton of Lexington, the attending physician, said that there must be still some mechanical lesion. I was confident that the symptoms were neurotic. The patient's condition became so alarming that I did not dare, on the strength of my own opinion, to persist in refusing operation. Dr. Stevens of Cambridge, who at first had looked upon the case as neurotic, became at last convinced of its mechanical origin, and advised operation. Even then, in my confidence at having at the time of operation examined digitally everything within easy reach of the gallbladder incision (stomach, pylorus, duodenum, and pancreas), I predicted a useless operation, only to find, in this misjudged woman, a *cancerous stricture* of the *jejunum* with almost complete obstruction! Then how clear became the connection between cause and effect; how characteristic of obstruction below the pylorus the symptoms—spasm in the epigastrium, pain, vomiting of bile, complete obstipation, with absence of distention and intestinal spasm! The wonder to me now is how I could possibly have failed to deduce the diagnosis of obstruction high up from such a history and physical examination. And yet how frequent is cancerous stricture of the jejunum? It is so extraordinarily rare that it would be ruled out of every eliminative diagnosis by its infrequency alone. Dr. Tilton was right; and the patient, after resection and suture, owes her life to his persistency.

Taking into account all the cases of biliary disease directly and indirectly dependent upon gallstones, including those in the pancreas and liver, pylorus and duodenum, including all cases in which a gallbladder origin has been diagnosticated, whether operated upon or not, and, moreover, considering, besides my own cases, those of my colleagues at the Massachusetts General Hospital—a total of at least fifteen hundred cases—certain facts become conspicuous:

(*a*) The grave dangers of biliary diseases depend, directly and indirectly, upon gallstones;

(*b*) The causes which lead to symptoms in cholelithiasis are always mechanical;

(*c*) Medical treatment, once gallstones begin to offend, is necessarily palliative, for it tends to show that in advanced and serious mechanical lesions treatment may diminish somewhat the severity of the symptoms—for example, a course at Carlsbad;

(*d*) Medical treatment has no effect whatever upon the stones themselves, either as preventive or as absorbent. Surgical treatment, when applied while stones are confined to the gallbladder, before impactions of any kind have taken place, is so safe that there is practically no mortality;

(*e*) At the moment the stone becomes grasped in an irritated and contracted gallbladder, or becomes engaged in the cystic duct, symptoms more or less severe, dependent upon pathological changes more or less extensive, begin. When, as the result of prolonged mechanical irritations and of infections, changes are extensive, and especially when these changes are in and about the hepatic and common ducts and in the pancreas, operations are more difficult, the dissections are deeper and in more hazardous relations, the patients are debilitated by long-continued symptoms. When patients are jaundiced, the dangers of operation are especially great and the mortality is large;

(*f*) The course of gallstones treated medically for symptoms of irritations and infections more or less severe, is toward a fatal end, through great suffering. In a few instances symptoms cease or are quiescent. Time is yet insufficient to show the ultimate results in many of these cases. In some, however, we have seen infections, perforations, permanent and fatal jaundice, secondary lesions of stomach, duodenum, liver, and pancreas. We have learned that Nature's methods are clumsy, dangerous, and inadequate.

That these propositions are sound, I have been convinced from over twenty years' experience. I do not at this time propose to prove their truth. Upon them, however, I base the conclusions of the present communication.

Assuming, however, the truth of the foregoing premises, the one conclusion, at least, is unavoidable, that gallstones should be removed in their simple and uncomplicated stages. What, then, is the most important consideration in connection with the treatment of patients suffering, directly or indirectly, from the effects of gallstones? Is it not the earliest possible diagnosis, when gallstones are in their simple and uncomplicated stages?

The diagnosis of gallstones when they first begin to offend, whether by the excruciating pain of a successful passage or by the grumbling discomfort of an apparent dyspepsia, is the first and most important consideration of all questions connected with the right upper quadrant. Unfortunately, the time when gallstones begin to offend is very uncertain. The symptoms of offending, too, are most difficult of recognition and interpretation. Typical and unmistakable gallstone colic—terrible, transitory, and recurrent—occurs in a small proportion of gallstone cases. Severe though the symptoms may be, their very severity and typical course point to slight pathological changes, and insure the safest of all gallstone operations. Persistent

and obscure symptoms, trivial and perhaps hardly noticeable, dependent upon the slow dislodgement and delivery of a single large stone, mean, from the nature of things, extensive pathological changes difficult of explanation and dangerous of repair. Indeed, if I were to make a rule, I should say that the typical and occasional biliary colics were more likely to indicate comparatively unchanged gallbladders than the obscure and persistent discomforts.

I am not advocating—at present, at least—that gallstones should always be removed when their presence is first suspected; for, while there are many considerations in favor of early explorations of the right upper quadrant, there are many against it. The chief objection to operations in this region is the difficulty of diagnosis,—I do not mean the difficulty—not to say the impossibility—of telling exactly what the matter is with the patient, but the difficulty of deciding with reasonable certainty that there is present *any* surgical lesion whatsoever. Moreover, when explorations of the right upper quadrant are freely advised for perhaps trivial symptoms and for symptoms which are not characteristic of any special disease, the door is opened wide for those indiscriminate, ill-considered, and unjustifiable surgical procedures, especially dangerous in the hands of the inexperienced, the amateur, the dilettante operator. Even when the diagnosis is plain, no one has a right to operate on the biliary tract unless he has had a large experience in general operating.

The diagnosis of diseases in the right upper quadrant is one of the most interesting and important chapters in internal medicine and in abdominal surgery. In many cases the diagnosis is so clear as to be beyond question; in others it is so obscure that it cannot be settled without exploration, although the symptoms may definitely point to one of three or four conditions. Indeed, in some cases great difficulty will be encountered, even with the hand in the abdomen

and with the parts fully exposed to light. In some instances, after the most painstaking research, the surgeon will reach an apparently conclusive diagnosis, and even then will be unable to find any lesion whatsoever. Nevertheless, the accuracy of the diagnostician in diseases of the pylorus, the duodenum, the pancreas, the liver, the gallbladder, and the right kidney is to me remarkable. The study of cases with lesions in this region is, to quote President Eliot, one of the greatest "joys of work." By numerous explorations in this area the surgeon is every day demonstrating the connection between cause and effect, between symptoms and pathological lesions. Whereas in the beginning no one could base a positive diagnosis of gallstones upon other than typical symptoms, to-day many a physician and internist makes, with the greatest accuracy, a diagnosis based exclusively upon the history, and particularly that part of the history which deals with pain in some form.

From the pathological point of view we find at the operating table four classes of cases: Cases in which changes are owing to (1) stones contained in the gallbladder itself; (2) impactions in the cystic duct; (3) impactions in the common duct; and (4) impactions in the hepatic duct.

Without going into details of the numerous pathological changes upon which symptoms in these various localities depend, it is sufficient to say that there is a very wide variation in these changes themselves and in their subjective manifestations. In the great majority of cases we are able to tell not only that a gallstone is present, but where it is situated; we can predict with accuracy what changes, if any, we shall find; what the difficulties in dissection will be; what the prognosis is as to life and as to health.

Jaundice as a symptom demands special mention. Its weight, when present, is great. Too much importance must not be given to its absence, however. Many practitioners to-day believe that jaundice is essential to the diag-

nosis of gallstones. In the great majority of cases jaundice is anatomically impossible, because the stones, situated in the gallbladder from beginning to end, cannot impede the biliary stream. It is a remarkable fact of recent demonstration in many of my cases that gallstones, sometimes in large numbers, may be situated in the hepatic and common ducts directly in the biliary stream without causing jaundice. Jaundice when gallstones are suspected, clinches the argument—makes the diagnosis positive; but its absence by no means eliminates the possibility of their presence.

The symptom usually relied upon for the diagnosis of gallstones is pain of some kind, with local signs in the region of the gallbladder. In those early cases in which the operative treatment is practically without mortality, almost the only symptom is pain. In the later stages also, when gallstones—through the mechanical changes which they cause—interfere with the biliary stream, pain is the first and most important symptom. Even when the changes are extensive, when the gallbladder can be felt, when there is jaundice, when there are signs of infection, pain is still the first and most important symptom. Pain is such an invariable and positive symptom of cholelithiasis that its absence, when all other symptoms point to gallstones, is conspicuous and suggestive. Indeed, the failure to get from the patient's history any account of pain, makes the diagnosis of gallstones improbable, and always favors the diagnosis of malignant disease either in the pancreas or elsewhere in or near the biliary tract.

In support of the proposition that gallstones should be removed as soon as they begin to offend, we must consider not only those trivial cases in which we can hardly fail to diagnosticate gallstones, but those in which the signs are obscure—the cases in which the symptoms may be described by what the layman calls "biliousness" or "dyspepsia,"—some form of pain or discomfort.

The pain of gallstones which, either at operations for their removal or at other operations, are demonstrated to exist, receives a great variety of names. I have interested myself in placing in parallel columns the patients' exact words in describing their discomforts, and the pathological lesions found. One sees a wide variety of descriptive symptoms—from what is roughly described as "biliousness" and "dyspepsia" to such expressions as "grinding pain," "deep soreness," "discomfort," "gastric distress," neuralgia, and neuritis. In a recent case the only symptom of gallstones that I was able to make out was a persistent "strenuous distress." In this case the patient for many years had refused operation. As a last resort the gallstones were removed, but the operation proved fatal.

It is much easier to ask questions than to answer them—it is much easier to ask what the symptoms of the earliest stages of gallstones are than to tell what they are. The two most important considerations in the diagnosis can be strongly emphasized. The first and most important is the pain itself; the second is the irregular recurrence of the pain, in a similar or in a gradually changing form. One attack of discomfort in the right hypochondrium may suggest gallstones; a second similar attack suggests them more strongly; a third makes the diagnosis probable; transitory tenderness in the gallbladder makes it almost certain; a transitory jaundice, though seldom present, establishes the diagnosis. The discomforts which gallstones in the gallbladder in their earliest stages, without mechanical impaction, cause, in the given case, are always the same. These discomforts are repeated at irregular intervals. They often seem to depend upon what is eaten, so that patients refer them to errors of diet. And yet the recurrence of symptoms at times when they cannot reasonably be said to depend upon anything eaten, favors a gallstone rather than a gastric origin. Irregularities as to frequency,

with irregularities as to time of day, add to pain and its variations the strongest argument in favor of gallstones. Periodicity as to time and regularity as to occurrence favor a gastric origin for pain. The relief of discomfort or of pain by food favors also a gastric cause. In simple gallstones no relief is possible except that which overcomes spasm.

Acute and chronic septic processes in the biliary tract have a symptomatology of their own dependent upon infections and their results. These are almost invariably preceded, however, by the symptoms of their causative factor, gallstones.

Whether gallstones suspended in the bile of a practically normal gallbladder ever cause symptoms, I am unable to say. I have found a single stone in a gallbladder not tense or distended, suspended in this fluid; and yet I have no doubt that this stone had at times become temporarily engaged at the cystic duct, by the contraction of the lower part of the gallbladder upon it. In such cases, when the stone becomes engaged, there may be a painful spasm, or there may be a discomfort, which is referred to by the patient as a dyspepsia or a gastralgia, or a biliousness, or an error in diet. Nevertheless, one cannot assume that gallstones in a comparatively unchanged gallbladder always cause symptoms, when occasionally, after operations which have demonstrated pathological changes, patients under the closest questioning have not been willing to admit the slightest pain or discomfort. I have seen gallbladders packed full of stones, without any bile, in cases in which the symptoms were trivial; and, as I have said, I have seen two cases in which there were gallstones large enough to obstruct the small intestine, and yet in which there were no symptoms.

Briefly, the diagnosis of gallstones in the early stages depends upon some form of pain recurring at irregular in-

tervals. The more violent the pain, the greater the probability of gallstones,—the very violence of the symptoms indicating a sudden impaction. The consulting surgeon, unfortunately, does not see the patient during these acute attacks. I cannot but think that in every instance the very first symptoms are due to contraction of the gallbladder upon the stones,—from which contraction, unsuccessful in expelling the stone, a spasm of the muscular fibres of the viscus must result. This spasm must either be perceptible at the time, or it must result in tenderness. A gallbladder may be excessively distended and yet be so situated as to be imperceptible; nevertheless tenderness to deep palpation will usually be expressed. It may, however, be tightly distended and yet cause no symptoms—subjective or objective.

With the attention of the practitioner drawn to the possibility of a gallstone origin in all obscure symptoms of the epigastrium and right upper quadrant, there will result, I think, a recognition of the early efforts of the gallbladder to rid itself of these foreign bodies. In addition to the subjective symptoms based upon pain or its modifications, some physical sign, either tenderness or distention, will suggest the lesion.

Before going farther into the symptomatology, let us pause a moment and consider what a positive diagnosis of gallstones in the early stages means to the patient, as contrasted with the diagnosis made in the advanced stages. In a total of perhaps four or five hundred cases of gallstones which I have myself operated upon, there have been no deaths in those early stages which I am now considering, when the stones are confined to a comparatively healthy gallbladder. When through impaction of the cystic duct changes have taken place in the gallbladder, changes owing to infection and obstruction, and especially when there has been gangrene of the gallbladder and local peritonitis,—

there has been a small mortality. The dangers of cholecystectomy, as compared with cholecystostomy, in clean cases, are small, but there is, nevertheless, in aseptic cholecystectomies, as compared with aseptic cholecystostomies, a mortality which cannot be disregarded. The pathological conditions which indicate total extirpation of the gallbladder increase the difficulties of dissection, prolong the operation, and, in the aggregate, therefore, necessarily augment the danger and the mortality. Acute infections of the gallbladder, with or without gangrene and perforation, though in my experience less fatal than similar infections of the appendix, have yet a considerable mortality. These cases which in their progress may sometimes be called fulminating, without prompt relief are through gangrene and perforation necessarily fatal. As in simple gallstone irritations, the success of operative treatment is in direct ratio with seasonableness of intervention. When thé stone has become impacted in the hepatic or in the common duct, we then have the far-reaching changes of obstructive jaundice, with increased difficulties of dissection, with failure in the patient's strength and in his powers of resistance. Here we have the great mortality of gallbladder surgery, by far the larger number of deaths in the surgery of the biliary tract are among these cases. In those malignant and hopeless changes due to long-continued gallstone irritation —illustrated by one of the cases briefly mentioned in the beginning of this paper — we have a strong argument in favor of the invariable removal of gallstones.

Reviewing thus briefly the brilliant results of the early operative cases and the large mortality in advanced gallstone disease, as surgeons see them, we cannot but be impressed by the desirability of recognizing at the earliest possible moment the presence of gallstones. It may not be wise in every case to remove them, — indeed I do not advocate the indiscriminate removal of gallstones known

to exist in the gallbladder. As I have said, I have not a few patients in whom I know gallstones to exist, and in whose cases I have not as yet advised operation. Knowing, however, to a certainty that stones are present in the gallbladder, and knowing also the precise anatomical conditions present, I am prepared to advise operation the moment the first symptom of impaction appears, unless this impaction should be of the briefest and most trivial character. A single recurrence of impactive (irritation) symptoms, like a recurrence of appendicular colic, is enough to justify operation. When gallstones are suspected, the indications for exploration are stronger than when they are known to exist, because, although we do not in the given case know the exact extent of the pathological changes in the biliary tract, we do know that in many cases these changes are out of all proportion to the symptoms upon which the explorations are based.

In making an early diagnosis of gallstones, one must consider very seriously the possible presence of those conditions of the stomach usually known as functional, for it would be as absurd to explore and drain the gallbladder for those transitory, permanent, or recurring discomforts which are usually spoken of as dyspepsia as it would be to perform a pyloroplasty or a gastro-enterostomy. Although it has been said that most gastric symptoms are in reality mechanical, I am not able to concur in this opinion. There are doubtless many cases treated as functional disturbances of the stomach or of the intestine, under the sweeping diagnosis of gastric, or, still worse, intestinal dyspepsia. So many patients come to my attention that are treated ineffectively for "intestinal dyspepsia" when the symptoms are really of mechanical origin, and amenable to mechanical treatment only, that I have felt called upon more than once to protest against a diagnosis so plausible, but at the same time so dangerous. So it is in the gastric symptoms

treated as dyspepsia. Dyspepsia is a symptom or a group of symptoms, in many cases without organic changes, but in many others with them. It is as dangerous a symptom —because so common and so little feared—as metrorrhagia or rectal hemorrhage, and should be watched and feared quite as much. The tendency to regard epigastric symptoms as functional and of little danger must be fought as much as the tendency to look upon metrorrhagia as menstrual or climacteric, and upon rectal hemorrhage as piles, —it must be fought even more strenuously, because serious organic lesions of the stomach grow so quickly beyond the possibility of surgical relief. It is a pity, too, that organic lesions of the stomach are so hard to recognize, as compared with those of the uterus and of the rectum, for the stomach anatomically and pathologically presents a field much more hopeful in the surgical treatment of malignant disease than either the uterus or the rectum.

On the other hand, it must not be forgotten that the larger proportion of gastric symptoms are really of functional origin. Few live to middle age without experiencing some of the symptons of indigestion—acute or chronic. True, many of these symptoms are owing to chronic appendicitis, gallstones, gastric or duodenal ulcer, and the like;—true, the majority are functional and of no surgical importance; yet chronicity, recurrence, and obstinacy are of such significance that these symptoms must be considered from the surgical point of view.

Symptoms which have been proved to be caused by gallstones, however little they may have conformed to the ordinary history of gallstones, cannot but suggest them when again encountered. Sudden distress in the epigastrium, therefore, however little inkling this symptom in itself may give of gallstones, becomes in the light of experience highly suggestive. In the light of reason and knowledge, however little epigastric pain and distress and other digestive symp-

toms may indicate a gallstone origin, they at least attract my attention to such a cause. Insensibly—unreasonably, if you will—these symptoms hint so strongly at a gallstone origin, that I have many times made a correct diagnosis upon them. And I have learned by long experience to follow, even more and more as the years go by, the lead of first impressions. Not that I would advise a beginner to do so; but what one has learned as to the value of his first impressions he should not too quickly forget.

The time at my disposal forbids a detailed consideration of the differential diagnosis of diseases of the right upper quadrant. It is a field prolific with interesting suggestions, a field which becomes the richer in possibilities of brilliant results the more it is explored. And upon what do these brilliant possibilities chiefly depend? First, upon an early diagnosis that mechanical lesions exist; and secondly, upon prompt mechanical relief.

The diagnosis in the early stages of most of the diseases which gallstones simulate lies in a correct interpretation of pain in some form or other. The most frequent of all the complaints that I used to hear at the enormous clinics of the Boston Dispensary, especially in the room for women, were those referred to the epigastrium, and treated by attention as far as possible to the diet. So it was in a general medical practice of twelve years. Had it been suggested at that time that many of these complaints were in reality due to mechanical causes, either in the alimentary or in the biliary passages, the incredulity with which the suggestion would have been received may possibly be imagined.

At the present time we know by large experience that many of these obstinate cases which to all general practitioners were—and are now—to the last degree discouraging under medical treatment, are as to cause purely mechanical and therefore remediable. That the pendulum has swung

too far in the direction of surgical treatment of these affections, many of us sincerely believe; but that the investigations of thousands of observers, both medical and surgical, will before long have settled pretty accurately these questions, I have no doubt. In the solution of them no one field can be more prolific of good results than that of diagnosis; and no more effective means of perfecting diagnosis can be found than surgical explorations which establish beyond question the relationship between the mechanical causes upon which the subjective symptoms depend and the subjective symptoms themselves.

The first differentiation in diseases of the epigastrium and right upper quadrant lies between the mechanical and the functional, between the surgical and the medical, between those which can be cured only by mechanical measures and those in which the parts concerned are in perfect mechanical condition, but in which the physiological processes are out of order.

This differentiation is possible only by the exclusion of mechanical causes of symptoms, and these, in turn, only by the symptoms themselves; for it is evident that mechanical causes which are not demonstrable by the senses must be shown to exist by subjective symptoms alone.

When, therefore, a tumor can be felt in the suspected viscus—whether gallbladder, liver, or stomach; kidney or pancreas or colon; when the stomach contents bear evidence of ulcer or cancer; when blood is demonstrable in the stools or cells of malignancy in the urine,—then the question of mechanical origin is easily settled. When, however, all external and demonstrable signs of organic diseases are absent; when the disease—if malignant of the pylorus for example—for mechanical cure demands the earliest possible recognition,—then the symptoms are necessarily subjective and the differentiation particularly difficult.

We will omit the consideration of perceptible physical

signs which make organic disease conspicuous and confine our research to those subjective symptoms upon which all early diagnosis and effective treatment depend. In some cases physical evidence may be at hand, even in the earliest stages of the disease, long before any subjective signs are present. We are not considering, I repeat, these glaring cases, but the obscure ones, those in which the disease is so young that surgery offers a reasonable chance.

The distinction between gallstones and gastric diseases is chiefly important because under a wrong diagnosis of organic disease, we may operate uselessly, as in simple dyspepsia, gastralgia, atony, gastritis, gastro-duodenal catarrh and the like. On the other hand, we may, under a wrong diagnosis of functional disturbance, withhold an imperative operation for gastric cancer, ulcer, strictured pylorus, or gallstones.

Practically, therefore, in the diseases of the stomach and gallbladder the diagnosis should aim first to eliminate the functional disturbances, and thus to avoid the unnecessary operation. Is it within the bounds of human knowledge thus to avoid error; or, in our zeal to operate early upon diseases, like cancer, which so soon become inoperable, must we expect a considerable percentage of unnecessary explorations? Will the greatest good to the greatest number result from a large number of operations in which a few will prove unnecessary, or from a small number of operations in which most will prove useless because too late?

The early symptoms of gallstones, as compared with those of gastric diseases, show pain in some form, uninfluenced by any of the causes which precede purely gastric attacks.

So prevalent is the impression that epigastric discomfort, distress, and pain are always owing to errors in diet, that the physician must be on his guard lest he himself be biased in favor of some dietary cause.

The distinction between gallstones and functional symp-

toms of the stomach lies in the patient's description of the pain; in its irregularity of recurrence; in the means required for its control; in the impossibility by any method of prophylaxis of preventing its return; in the absence of constitutional effects; in the maintenance of nutrition; and in the similarity of succeeding attacks with perhaps a gradually progressive change in them as the pathological conditions causing the symptoms themselves change.

Gallstone diseases, at whatever stage, are at times distinguished from early organic changes in the stomach only with the greatest difficulty. We cannot expect to tell the difference until there are some symptoms at least of organic disease of the stomach. Indeed, herein lies the real difficulty of successful gastric surgery. An organic gastric origin of epigastric symptoms means an organic change of such extent that physical signs of some kind are usually evident. To take a single symptom: recurrent spasms of pain in the epigastrium dependent upon a pyloric stenosis, though they may suggest gallstones at first, later, by their very persistence, make a gallstone origin impossible. The gastric spasm becomes in many cases visible, with vomiting, emaciation, and signs of dilatation. Until the stenosis becomes great enough to cause gastric spasm, there will be no pain, except that of the pain causing stenosis. Here again the importance of irregularity as to time becomes evident. Gallbladder spasm may at first suggest gastric spasm, but time will soon eliminate most positively a gastric cause of pain. Strictly speaking, however, pyloric stenosis should not be considered, because we are considering conditions without physical signs, and stenosis cannot but cause perceptible physical signs—gastric spasms.

We are brought again, therefore, to consider the cause and nature of epigastric pain as dependent upon organic causes imperceptible to the senses. Here we must rely

upon the evidence of those observers who have studied the connection between pain as described by the patient and the anatomical findings of the operation. I do not know myself where to find them described; for the time has not come, in my experience at least, when I have felt justified in opening the epigastrium for symptoms which do not suggest very strongly either gallstones or some mechanical lesion of the stomach. That the symptoms in the epigastrium described to me do suggest correctly gallstones, is shown by the almost invariable presence of gallstones.

What, then, are the symptoms of gastric disease, both functional and organic, which in my experience do not mean gallstones? They are various forms of pain dependent unmistakably upon diet,—pain which occurs with regular reference to certain periods of digestion; which always follows the taking of certain foods; which persists a long time; which impairs strength and reduces weight if only temporarily; which, finally, does not require heroic remedies for immediate control.

Functional gastric troubles are eliminated by their response to medical treatment, whereas organic are confirmed by the failure of that treatment.

It may be inferred that pain and its various manifestations are caused by functional derangements of the stomach when they are brief, regular, and easily controlled by treatment based upon the diagnosis of functional disease; when the symptoms for years have appeared at intervals without serious effect upon the constitution, and when there has never been at any time a suggestion of biliary obstruction; or when in the region of the gallbladder there has never been a localized tenderness or a perceptible tumor.

Organic disease of the stomach—if an ulcer—shows itself early by almost unmistakable signs. Confusion between gallstones and ulcer need never exist, except perhaps in contrasting the early stages of ulcer with the late stages

of gallstones. Age, hæmatemesis, color of skin, length of attacks, and methods of control in ulcer, contrasted with age, jaundice, length of attacks, and methods of control in gallstones, permit few mistakes.

In the late effects of ulcer there is much more liability to confusion, in distinguishing between chronic cholecystitis with gallstones, and chronic pyloric obstruction, with gastric dilatation. It will usually be evident, however, in which case to expect the extensive changes of gallstones and in which those of gastric obstruction and food decomposition.

But we are considering, I find, the cases in which there is extensive and visible evidence of disease, though not perhaps conspicuous evidence. Can we make a distinction between gallstones, functional gastric symptoms, and beginning ulcer or beginning cancer? This is the real question.

I will try to answer it by asking another: Does cancer of the stomach in its inception cause pain, or, in fact, any other symptom whatsoever? If it does, what is the nature of this pain and of those other symptoms? If it does not, the early diagnosis of cancer is impossible.

I have discovered cancer of the alimentary tract in patients who have never shown the least sign of that disease. No form of cancer can cause such suffering as cancer of the intestines, and yet in these cases there is no pain before obstruction begins. I am convinced that cancer of the pylorus, of the body of the stomach, is absolutely without symptoms in the early stages,—before there is mechanical obstruction or pressure, before there is ulceration and hæmorrhage, or before the gastric wall is so involved as to interfere seriously with digestion through changes in the gastric juice.

In a word, gastric cancer, at the stage most favorable for operation and permanent cure, cannot be diagnosticated

unless, fortunately, it ulcerates early over a distinctly localized surface, or unless it happens in the proliferative, warty, or papillomatous stage to fill and obstruct the pylorus. Gastric cancer is like cancer of the breast and cancer of the uterine cervix—painless in its early stages.

It follows that the distinction between ulcer and cancer of the stomach in those cases in which there is only pain, and gallstones in which there is only pain, must be based upon the character of the pain itself; its situation and extent; its time of occurrence; its direction, continuity, persistence, recurrence; its sharpness, dulness, and like chacteristics; its exciting causes and its controlling treatment; its constitutional effects, its periodicity, and, now and then, under careful and persistent observation, some objective symptom so trivial and doubtful perhaps as to be almost classed as subjective. All these things will tend to prove the existence. either of a biliary or a gastric cause which demands surgical exploration. In many cases this evidence will point so conclusively toward the biliary tract as to exclude the gastric. Indeed, organic gastric diseases are best diagnosticated by the exclusion first of biliary.

In all pancreatic lesions which simulate or depend upon gallstones the only thing of importance in diagnosis is the elimination of the cases in which operation is useless. Can any inoperable pancreatic lesion be eliminated from the diagnosis with sufficient positiveness to justify non-intervention on the ground of absolute hopelessness? I am sure that it cannot.

Cancer of the pancreas, a common disease, is hopeless, but it cannot be distinguished beyond a doubt unless there is a tumor so perceptible as to be accurately palpated. Such a tumor may sometimes exist, but I have never seen one. Pancreatic cancer is a possibility to be considered in the indications for operation and in the prognosis whenever exploration of the biliary tract is proposed. The diagnosis

of pancreatic cancer is nevertheless easy, for it depends upon painless jaundice and emaciation. Evidence in the stools, whatever its theoretical value, has but little practical importance. The chief element in the history in favor of malignancy is the absence of pain from the very beginning of the jaundice, and perhaps long before the first appearance of jaundice; whereas if the jaundice were owing to gallstones, we should expect, in the progress of the stone from the gallbladder to complete impaction in the ducts, pain more or less severe. No diagnosis of pancreatic cancer should be made until there have been the most searching inquiries as to the previous occurrence of pain in some of the forms described—whether the diagnosis is gallstones, dyspepsia, biliousness, or something else. In certain cases the surgeon will find an impacted gallstone even when the history is absolutely without a suggestion of previous pain. In some cases the pancreatic portion of the common duct will contain a stone which, through the pancreatic tissues, will feel like a malignant nodule. The diagnosis of this condition beforehand is impossible; and so is the diagnosis of a stone in the opening of the canal of Wirsung, where I once found one (Vol. LXI., p. 174).

Chronic pancreatitis may be expected in a certain proportion of cases especially when there is jaundice. The diagnosis of a gallstone cause for jaundice does not exclude this condition, for it accompanies or follows gallstones, whether in the gallbladder or in the cystic, hepatic, or common duct. I have found enlargement of the head of the pancreas, without gallstones, in the comparatively young, and the disease has been permanently cured, apparently, by cholecystostomy. In suspected acute lesions of the pancreas a history of gallstones adds to the strength of the diagnosis. In all pancreatic lesions, except cancer, the exploration which establishes the diagnosis may be followed by curative operation, especially cholecystostomy for chronic pancreatitis.

The diagnosis between gallstones and certain lesions of the liver is interesting; but hepatic diseases are so rare that the question of differential diagnosis between these two conditions seldom comes up.

During my last service at the Massachusetts General Hospital a patient was transferred to my care who had been operated upon for suspected gallstones. None were found; but on the anterior surface of the great lobe of the liver there was a rounded, smooth prominence which suggested to the operator a neoplasm, probably malignant. Cholecystostomy was performed to relieve the existing jaundice. There was much hæmorrhage following this operation, but the patient survived. For several weeks I watched the case, trying to decide upon the diagnosis and the best treatment. Before I had decided, however, the patient began to bleed from the nose, and she died of uncontrollable hæmorrhage. The autopsy showed a hypertrophic cirrhosis, which no physician or surgeon had for a moment suspected.

Abnormal liver conditions, whether accompanied by jaundice or not, sometimes suggest gallstones and are operated on uselessly. Can these diseases be successfully eliminated in making the diagnosis? I do not think that they always can. I have found gallstones in a hard, irregular tumor which was supposed to be a mass of cancer, but which was really an enlarged, irregular, and thickened gallbladder. In other cases the diagnosis of gallstones has proved correct, but there has been also malignant disease.

Cancer of the liver, not associated with gallstones, is eliminated by its painlessness and by the character of its jaundice, as there is jaundice from obstruction of bile in one part of the liver, and bile in the stools from another part. Liver abscess might be confused with an acute cholecystitis were the abscess in the immediate vicinity of the gallbladder. Such a condition would, however, be so rare that it need not be seriously considered.

Perhaps the most important disease to eliminate is cirrhosis with jaundice. This condition, however, is extremely unusual in a form which even remotely suggests gallstones; an hypertrophic cirrhosis might; no cirrhosis with ascites would, for the presence of ascites would immediately rule out a gallstone origin.

Many errors in diagnosis in diseases of the right upper quadrant come from inadequate study. Except in the plainest cases the patient in whom gallstones are strongly suspected should be examined several times, if the symptoms are not urgent. If the case is obscure, much time should be spent upon it, and the prognosis should be guarded. Cases of obscure origin, however, in which the symptoms are severe, must be explored at the earliest possible moment, even under the possibilities of an unnecessary operation.

Next in importance to the establishment of the diagnosis, especially with reference to gallstones, are the indications for treatment.

In many diseases the indications for treatment are comprised in the disease itself. In gallstones, for example, once the diagnosis is made from the symptoms that they cause, operation in the absence of contra-indications is always advisable. These contra-indications are the existence of serious lesions of other important organs, cachexia, anæmia, hæmophilia, inadequate powers of resistance from other causes, and perhaps the habit of tardy convalescence from trivial diseases—of falling an easy prey to evil complications.

Shall we operate when gallstones which cause no symptoms have been demonstrated at other operations? Are exploratory operations indicated when gallstones are only suspected? Gallstones which are known to exist should be removed the moment symptoms dependent upon them appear. I personally believe that removal is wise in all cases,

whether they offend or not, provided the patient is in the best of conditions; if he is not in perfect condition, I should await developments. In cases in which gallstones are suspected, we should explore if the suspicion lies between gallstones and other conditions as serious or more serious. When, for example, the diagnosis lies between gallstones and a simple dyspepsia, the benefit of the doubt would consist in postponing exploration; when the diagnosis lies between gallstones and cancer of the pylorus, the benefit of the doubt demands immediate exploration.

The rule by which I have been guided and which I have rarely if ever had occasion to regret is that when symptoms exist in the right upper quadrant which are of a chronic character, when the attacks of pain or distress continue especially at irregular intervals, and when the attacks of pain in any form suggest the presence of gallstones, exploration is indicated, particularly when palliative measures have been employed in vain for a reasonable length of time. When later signs confirm the existence of gallstones, any measures other than surgical are unreasonable and ineffectual.

DISCUSSION.

Dr. R. H. Fitz, of Boston: Despite the recognized importance of the liver in promoting digestion, maintaining nutrition and acting as a safeguard against certain poisons, it must be admitted that our means of determining a failure in its various functions are extremely limited. There is but little difficulty in making a diagnosis of probable gall stones, and possible complications, requiring active, and more or less immediate mechanical treatment. More important is the diagnosis of the conditions which lead to the formation of gall stones with a view to their prevention. For generations such terms as congestion, torpor and sluggishness of the liver have been in common use, but the well-informed physician recognizes that they are merely conventional, and are by no means indicative of demonstra-

ble disturbances of function of this organ. The liver thus is made the scape-goat for what frequently must be regarded as a faulty action of the stomach or bowels, too often induced by excesses or negligence on the part of the sufferer. Indeed, the remedies which are given with the announced purpose of acting on the liver, if they do not produce a more or less energetic cartharsis are considered to have failed in their effort to produce "portal depletion."

At present, as in the past, the clinician has no other means of satisfactorily determining an alteration of the functions of the liver than by an inquiry into the greater or less ease with which the bile leaves the biliary tract. He is unable to recognize alterations in the quantity or quality secreted under the influence of disease, and it is impossible for him to determine the state of other functions of the liver under morbid influences.

The same holds true in great measure with regard to the clinical determination of functional disturbances of the pancreas. These were made the subject of an address before the Congress of American Physicians and Surgeons a few years ago, and an attempt was made to bring forward what had been done in this direction, and to outline what was in process of investigation. It is unnecessary to state that the work of the intervening period has served no other purpose than to show that the expectations of the past have not been fulfilled, and that no material advance has been made in the clinical recognition of disturbances in the function of this organ.

It is the province of the future to throw light upon these questions. They are in the first instances problems for the laboratory, and the splendid provision made for such researches in the new buildings of the Harvard Medical School are indication of their importance. It is of especial significance that a new laboratory is to be added to the series, one for the study of Clinical Pathology, in which the investigation of matters of importance in the practice of medicine shall be paramount.

The diagnosis of diseases of the liver and pancreas, therefore, must depend now, as heretofore, upon the determination of the position, shape, density, mobility and sensitiveness of the parts concerned in addition to the data derived from the history of the patient.

With the admission of the impossibility of making an accurate clinical diagnosis of the disturbances of function of the liver and pancreas, comes the simplification of treatment. To attempt to stimulate or to depress assumed disturbances in the function or functions of the liver incapable of recognition is a leap into the dark. It is far safer to undertake the treatment only of those disturbances connected with obstruction to the passage of bile and pancreatic juice into the intestine, with suppurative and necrotic areas, and with calculi and tumors. It is the mechanical treatment of mechanical disturbance which is fruitful at present, while the functional treatment of disturbances of function is the task of the future. It is left for the physician to aid in the determination of the time when such mechanical measures shall be applied, either through his recognition of the failure of diet and drugs to afford relief, or by the appreciation of persistent evidence of the maintenance of nutrition despite the increasing size of the tumor. With the exception, perhaps, of certain syphilitic lesions of the liver and pancreas it must be admitted that the independent diseases of these organs of a serious nature, if not remediable by surgery, are incapable of cure by any other method of treatment.

Dr. S. J. Mixter, of Boston: The subject is such a tremendous one that in a discussion there is little time to lay stress on more than a very few of the points that occur to the speaker.

First, I want to say one word as to diagnosis,—the importance of recognizing the fact that recurrent attacks of pain in the region of the liver almost always mean gall stones. If one goes on that idea, and with a history of frequent recurrent attacks of pain makes an exploration, it is very seldom that he goes wrong. He may not find gall stones, but he will find something that produces this condition.

There is one condition which we find instead of gall stones, which produces similar symptoms, and that is a chronic inflammation of the gall bladder.

I think it is the duty of the surgeon also, in the way of diagnosis, in all cases of laparotomy, except in very young people, where the incision is large enough, where the pa-

tient's condition is such as to warrant and where there is no dealing with a septic condition, to examine the gall bladder with the hand. I also think it is his duty to remove gall stones when they are found if the patient's condition is such as to warrant it. This procedure is simple, and harmless, and I think should always be done, but it should be done in a proper manner. In certain cases, as Dr. Richardson has said, you know there are gall stones present, and you do not remove them. I have done hysterectomies on cases where the patients had in their gall bladders gall stones of consiberable size or number, but for some reason or other their condition at the time was such that it was considered best not to remove the stones, but with those patients, any symptoms appearing later can be acted upon immediately.

It must be recognized, however, that the most serious of all the gall bladder diseases are those which depend on acute inflammation without the presence of gall stones. Some are due to the presence of gall stones, but they are absent in some where there is a severe inflammation. We see such in typhoid fever.

A patient was brought into the hospital without any history, without any friends, a very sick man, with high temperature. I discovered a tumor in the region of the gall bladder. I operated and found an enormously distended gall bladder filled with pus, and drained it. The man died and we found that he had a severe typhoid fever. Some of my friends found fault for operating upon a case of typhoid fever.

The question of choice of operation has not been spoken of, and I think that is something which is important, but about which surgeons differ to a certain extent.

In gall bladder diseases, whether due to stones or to chronic or acute inflammation, very often the question comes up, Shall we simply drain, or shall we excise? Now I think this depends upon the experience of the surgeon. There is, as the Mayos have shown, a slight added risk in excision of the gall bladder. To my mind there is an added risk in leaving an inflamed gall gladder, that is, one with chronic inflammation. I think it is better to excise rather than to drain. I have done this without any ill effects. It

is a very easy operation, and even where the ducts have to be drained it is not difficult to drain them after the gall bladder is excised.

One point I think should be emphasized, and that is, in suspected gall stone cases, in inflammation of the gall bladder, the safest thing is exploration, because the after effects of very many cases of gall stones are so disastrous that it is better occasionally to open the abdomen unnecessarily than to leave the case to go on to impaction and ulceration which are so very much more fatal than the simple opening of an early case.

Dr. F. B. HARRINGTON, of Boston: This subject is full of unanswered questions. We are coming to feel more and more that operation is called for when stones are known to exist or are strongly suspected.

There are two pictures of the operative conditions in gall stone disease which we should remember. First the simple one of the presence of gall stones without sepsis. Here we have a condition analogous to the simple quiescent appendix in which operation is safe and the certainty of permanent cure is great. This condition if not relieved by operation may lead and often does lead to the second picture, the septic condition of the bile tract or the bile infected condition of the blood and tissues. These conditions may be combined, but alone or together they produce situations of the greatest gravity. I would therefore advise early operation in gall stone disease. As has been said, many cases of chronic indigestion are the result of the presence of gall stones. An interesting question is what is to be done with the gall bladder. It should be removed if much thickened, if gangrenous, or if there is a possibility of a tumor. It should be removed if there is a stone low down in the cystic duct, since the removal of this stone is likely to be followed by a stricture and obstruction of the cystic duct and pain from distention of the gall bladder by mucous secretion.

I formerly believed that the gall bladder, when once it had shown a tendency to develop gall stones, should be removed. I have changed my opinion because stones never or rarely form a second time, and because the removal of the gall bladder is a slightly more dangerous operation than

its drainage. Moreover drainage of the gall bladder is simpler and safer than drainage of the common or hepatic duct. It should, however, be remembered that cancer is sometimes primary in the gall bladder. The Mayos report one case of cure of cancer following early removal of the gall bladder.

ARTICLE XIX.

THE ANNUAL DISCOURSE.

THE SEGREGATION OF CONSUMPTIVES.

By J. F. ALLEYNE ADAMS, M.D.
OF PITTSFIELD.

DELIVERED JUNE 12, 1907.

NOTE.—At an Adjourned Meeting of The Massachusetts Medical Society, held Oct. 3, 1860, it was

Resolved, "That The Massachusetts Medical Society hereby declares that it does not consider itself as having endorsed or censured the opinions in former published Annual Discourses, nor will it hold itself responsible for any opinions or sentiments advanced in any future similar discourses."

Resolved, "That the Committee On Publications be directed to print a statement to that effect at the commencement of each Annual Discourse which may hereafter be published."

THE SEGREGATION OF CONSUMPTIVES.

Mr. President and Fellows of The Massachusetts Medical Society:—

The physician's path, though a good one to follow, is neither smooth nor easy. His life is a warfare, from the diploma to the grave—a warfare demanding the constant use of all his powers, but whose visible rewards are not commensurate with his expenditure of force. He strives, not for himself, but for others, and others enjoy the fruits of his victories. But this fact does not deter men from entering our profession. Rather does it tend to draw into it those who wish to lead fruitful lives, and it gives to the medical vocation a satisfaction which is beyond all material returns. The physician is not a soldier of fortune, seeking only gold and glory, but a crusader enlisted in the army of God, and fighting for the rescue of suffering humanity. This is the crowning glory of the doctor's life, and it is this unselfishness which ennobles his profession, and gives him a great and peculiar influence—an influence which is generally recognized by the community and which brings him great opportunities for usefulness.

Thus it comes about that a medical practitioner is, to a greater or less extent, a missionary. He is always finding and doing some work, unpaid or underpaid, for his fellowmen, because his training and his position render such work possible and natural. In freely serving the poor, in their homes or in hospitals, his opportunities for good are not

exhausted. In all philanthropic measures he is to be relied upon as a helper, and in those directly pertaining to the public health, he is naturally a leader. He has unequalled facilities for disseminating knowledge, for awakening interest and for guiding benevolence. The evidences of this influence are to be seen on every hand. Have you not all exerted it? and have not your laborious lives been brightened and sweetened by your success?

If the influence of the physician in his individual capacity is so great, how much greater must be that of the profession as a whole! When this acts as a unit great things can be accomplished, and our medical societies render such action possible. This organized force has done good work in the past: it has persistently opposed error with truth, ignorance with knowledge, charlatanry with science; it has given to the world the benefit of those marvellous discoveries of the past century, which have prevented untold suffering and rescued millions from death; and it stands ever ready to champion every good cause whose object is to save human lives and make them better worth living.

It is therefore the thing to be expected that we, Fellows of The Massachusetts Medical Society, on this our anniversary, should ask ourselves and each other, "What is the cause which this year most urgently demands our help?" I think you will agree with me that the one paramount problem which to-day confronts us is the suppression of tuberculosis, and that this is the cause which now calls most loudly for our united and hearty support.

The knowledge that it is possible to exterminate this scourge of our race has deeply stirred the minds of men—and flooded the world with the light of a new hope. A mighty war is now in progress—a war which is uniting under a common banner people of every race, color, language, nationality, class, party and creed. Already have notable victories been won, but we are still in the early stages of

the campaign, and it will require years of valiant and united onslaught to rob tuberculosis of its terrors. Meantime, the people look to us for leadership: and it is incumbent upon us to consider, year by year, what battalions are to be organized, what engines of war constructed and what tribute levied upon the community for the successful conduct of the campaign.

As a contribution to the discussion of this subject, I will ask you to consider with me that one of its branches which seems to me of the highest importance—namely, the prevention of consumption by the separation of the sick from the well.

Since pulmonary tuberculosis is known to be actively infectious, through a specific germ, it is perfectly evident that the isolation of all infected persons would, in a few years, stamp it out. It is the leprosy of our time; and, as leprosy is kept under control by the segregation of lepers, so may consumption be eradicated by the segregation of consumptives.

Let us suppose all the power in our country to be centered in a despot of unlimited power and wealth, and also filled with a burning desire to confer great blessings upon his subjects. Such an one might say "Consumption is the greatest enemy of my people. I will destroy it, and that quickly." Could he do it? He could, and how? He would order the formation of many great camps, or enclosures, where all consumptives would be forcibly colonized. These would be placed not far from the cities and towns, but far enough to find pure air and sunshine. There would be tents, cabins or shacks for the incipient cases, and hospitals for the advanced. There would be ample space and much freedom within the camp, but no passing the bounds. Here many early cases and some of the advanced ones would be restored to health and be permitted to return to their homes, while those who must die would have their illness bright-

ened by good care and every possible alleviation. By this removal of consumptives, infection would cease, and since the patients would no longer leave the disease as a legacy to their families, new cases would grow fewer and fewer. The change in ten years would be marvelous, and if our supposed despot should live to the good old age he would deserve, he would see his camps nearly deserted and consumption dwindling to an insignificant place in the mortality tables. At the end of his benificent reign, his grateful people would erect a monument inscribed with these glorious words "To the Conqueror of Consumption."

The day of despots has now gone by, but the power which they wielded still remains, vested in the people, and the people are able to accomplish this same result. When once they are educated up to the idea, when they fully understand that the thing can be done and that the doing of it will save multitudes of lives and remove one of the chief causes of suffering, poverty and sorrow, will there not be a great popular uprising, when the people themselves will demand the enforcement of this act of despotism, which will separate the infected from the uninfected, to the end that the plague may be stayed? They will be a little longer about it than our despot would have been—measures too arbitrary and violent might engender sedition and wreck the treasury,—and they will try to bring about the desired result without too much restraint of personal liberty and without danger of financial disaster, but in good time this thing will be done. It is safe to prophesy that the days of tuberculosis are numbered and that future generations looking back upon our time will say, "There were giants in those days, for they destroyed that arch-enemy consumption."

Through the untiring efforts of a great number of medical men and some very earnest laymen, the people are being taught the value of local associations, of medical inspec-

tion, of visiting nurses, of anti-spitting regulations, of disinfection of houses, of registration of cases, of out-door life, of dispensaries and of sanatoria for incipient cases. But the most urgent need of all, the hospital for advanced cases, is not yet sufficiently recognized. It is not a philanthropic interest in the patients themselves which is the chief reason for building these hospitals, though this would be reason enough; but there is a far more weighty one—the prevention of infection, whereby alone the endemic may be arrested. The public need be shown the necessity for such hospitals, and state, city and town governments should be brought to realize that the appropriation of public funds for this purpose is a patriotic duty on grounds, not only of humanity, but of self-preservation. The vast expense, however, will be an almost insuperable obstacle to the sufficient carrying out of the plan. But it is a war measure, and we must learn to consider it as such. "Millions for defence" are not begrudged, against human enemies; and why should they be against the far more hostile and bloodthirsty bacilli? But, for the relief of the overburdened tax-payers, we need also to enlist the interest of private philanthropy. Persons of wealth should be shown that in no way can they use their means to help their brother-men more wisely and efficiently than by building and maintaining these hospitals for consumptives. This needs to become the great popular charity.

The State of Massachusetts, which built at Rutland the first state sanatorium for incipient cases, is wisely undertaking to provide also for advanced cases. A "Commission to investigate measures for the relief of consumptives" was appointed a year ago, and presented its report last March. So admirably was this report prepared, so wise were its suggestions and so convincing were its arguments, that already has the legislature passed bills carrying into effect nearly all of its recommendations. These bills provide, 1st,

for the immediate construction of three Infirmaries for tubercular patients, of 150 beds each; 2d, for the appointment of State Inspectors of Health; 3d, for compulsory registration of cases; and 4th, for giving greater effect to the anti-spitting law. Surely this is a remarkable result. A Commission which has brought about the enactment, in a single session, of so many beneficent laws, is entitled to our gratitude and admiration. Three of its six members are Fellows of The Massachusetts Medical Society, and it is fitting that their names should be mentioned and remembered. They are Dr. H. P. Walcott, Chairman of the Commission and Chairman of the State Board of Health, Dr. A. S. MacKnight of Fall River, and Dr. C. S. Millett of Brockton, Secretary of the Commission. Their lay associates are Messrs. Brackett, Adams and Porter.

To this roll of honor should be added the name of Senator Frank M. Chace of Fall River, who had charge of the "Three Sanatoria" bill of Ex-Representative Googins of Cambridge, who for four years from 1901 to 1904 tried to secure the passage of a similar bill, and of Senator Frank G. Wheatley, M.D., another Fellow of our Society, who had charge of the bill providing for State Inspectors of Health.

There are many others who have been active in securing this beneficent legislation, but I will here add but one other name, that of Governor Curtis Guild, Jr., who in his last inaugural recommended measures for the relief of consumptives, and who has done all in his power to advance it.

This Commission undertook a census of consumptives in Massachusetts and obtained from physicians reports of 7779 cases, of which about one third are classed as incipient. But, as the Secretary of the Commission stated to a committee of the legislature, the actual number of cases is probably double these figures, which seems extremely likely, when we consider that there are 5000 deaths annually. It is quite safe to place the number of Consumptives in the

State at 15000, of whom 5000, or less, are incipient, and 10,000, or more, advanced. Let us inquire how nearly the three new infirmaries will come to accommodating this number.

We have already, in the various institutions of the State, about 400 beds for incipient cases and 500 for advanced—making 900 in all. The 450 beds in the three new infirmaries will make 1350. Each bed will be sufficient for two or perhaps three patients a year. If we say three patients a year, the 1350 beds will provide for 4000, leaving 11,000, or nearly three quarters, unprovided for.

The building of these infirmaries, therefore, is not going to be enough to stamp out the disease, but it is a long step in the right direction, it will afford much relief and will be an object lesson to demonstrate their value and the necessity for increasing their number in the future.

It will not take long to show the value of this separation of advanced cases. When we remove them from their homes and from public places, the number of incipient cases will begin to fall off. Every person with advanced tuberculosis is likely to infect at least one other person, so that under present conditions there is no possibility of the disease dying out.

The long continuance of the disease renders impossible such house quarantine as is enforced in the case of smallpox, scarlet fever, measles and diphtheria. However careful may be the patient and his family, there is always danger that, through some relaxation of the rules, germs may be conveyed to another person. Such accidents may happen through forgetfulness, weariness, excitement, intemperance or delirium. In the last stages of the disease, the patient often expectorates at random all over the bed, the floor, and even the walls, thus endangering the wife or daughter who is in attendance, other members of the family who pass through the room, and children who may be

creeping upon the floor. Such cases ought not to die at home. They should be moved to a hospital, where they can have more comforts and not be sowing the seeds of disease among those whom they love best. This separation will be resisted at first, on sentimental grounds, but the arguments for it are so strong that resistance will lessen with time. Especially will this be the case if these hospitals are not placed at such a distance as to forbid frequent visits from members of the family. These visits, which can be made without danger, will relieve homesickness, and make the separation less complete and depressing than it would be if the patient had to be removed far from home.

The sanatoria for incipient cases are doing a great work, restoring to health a considerable number of persons who would otherwise die. At Rutland during the eight years since the Sanatorium was opened, the number of cases discharged "arrested or apparently cured" has been 1487, or over 41 per cent. of all cases treated. This is a handsome showing; it justifies the outlay for the Sanatorium, and is full of encouragement for the future; but, compared with the number of consumptives in the State, the figures are not large. It has been the policy of the Sanatorium to restrict it to incipient cases, and such are never refused, but hitherto there have not been enough such applicants to fill the beds, and rather than to let the beds remain empty, a sufficient number of moderately advanced cases to fill them have been received. At present, the number of incipient applications has increased to such an extent that few advanced cases can be received; and the question every day arises, What can these people do? The doors of Rutland are closed against them, and properly so, for there incipient cases have the prior claim; but their need is great, and there is, as yet, no place for them to go.

The number of rejected applicants last year was about a thousand, and if we add to these nearly 400 more, who,

after a few weeks or months, were found not to respond to the treatment and were discharged, these alone are sufficient to fill the three new hospitals which are now proposed. This proves the urgent necessity for their speedy construction.

In Massachusetts, the provision for incipient cases at present consists of the State Sanatorium at Rutland with 350 beds, and two private Sanatoria—that of Dr. Bowditch at Sharon, and that of Dr. Millet at East Bridgewater, each with about 20 beds.

For advanced cases, about 250 beds, or half of all those in the State, are in Boston institutions, namely:—

The Channing Home.
House of the Good Samaritan.
The Cullis Consumptive Home.
The Free Home for Consumptives.
St. Monica's Home for Colored Women and Children.
The Boston Almshouse and Hospital.
Carney Hospital.

Outside of Boston, the principal provision is at the Tewksbury State Hospital, which has 140 beds for consumptives, besides a camp of 36 beds. The other institutions, where advanced cases are received, are:—

The Holy Ghost Hospital for Incurables, Cambridge.
Lowell City Hospital.
Fall River City Hospital.
Several private hospitals in Rutland.
Miss Sullivan's private hospital, Pittsfield.

A Sanatorium for Consumptive Prisoners is also just completed at Rutland, and the Danvers Insane Asylum has separate cottages.

To this list may be added the Mattapan Day Sanatorium, maintained during the warm months by the Boston Society for the Relief and Control of Tuberculosis, and the

Day Sanatorium of the Lowell General Hospital. A certain number of consumptives are also to be found in Insane Hospitals, almshouses and prisons. The General Hospitals, as a rule, receive such only in exceptional cases.

Boston is soon to have a large addition to its facilities in the Consumptives' Hospital now about to be built at Mattapan. In establishing this hospital this city has set an example which every other city will do wisely to follow.

Besides what is being accomplished by the sanatoria and hospitals, much good work is being done by dispensaries and clinics. Among these are to be specially mentioned the tuberculosis clinic of the Boston Dispensary, conducted by Dr. Otis; the Out-Patient departments of the Massachusetts General Hospital, Boston City Hospital and Carney Hospital, the Berkeley Infirmary and Trinity Dispensary. The tuberculosis classes have also proved extremely useful, and are well worth study and imitation. These are "for the home treatment by sanatorium methods of incipient and moderately advanced cases." Such are the Emmanuel Church Tuberculosis Class, conducted by Dr. Joseph H. Pratt; Arlington Street Church Tuberculosis Class, by Dr. N. K. Wood; Suburban Tuberculosis Classes, by Dr. John B. Hawes, and the Cambridge Tuberculosis Classes.

The Boston Association for the Relief and Control of Tuberculosis, and similar organizations in Cambridge, Worcester, Springfield and Brockton have also become important factors in this great and many-sided enterprise.

By these various means, tuberculous patients are treated in their homes, are instructed in the methods of cure and prevention, receive the care of physicians and visiting nurses, some receive aid, and some are enabled to go on with their usual occupations.

All of this work is of great and far-reaching importance, but would be made infinitely easier and more effective if

the hospital accommodations were sufficient for those cases in which the home conditions are unfavorable. That these conditions are unfavorable, in a large proportion of cases, we all know, and we also know how utterly inadequate are our 500 beds to the needs of the 10,000 advanced cases in the State.

In other states, the lack of provision for advanced cases is as great as in our own, and in most cases greater. For incipient cases, provision is becoming abundant. The sanatorium method, which was originated by Brehmer, in Germany, in 1859, and which was first put into operation in this country by Trudeau, at Saranac Lake, in 1885, has been attended with such wonderful results that these institutions have rapidly multiplied. They are now to be found in nearly all of the northern states, New York standing at the head of the list, and the majority being private institutions. But the example of Massachusetts in building a state sanatorium has been followed, and there are now 14 state sanatoria completed or building, besides 3 others which receive state appropriations. The United States government has established sanatoria—at Fort Bayard, N. M., for the Army, at Fort Stanton, N. M., for the Public Health and Marine Hospital service, and at Fort Lyon, Col., for the Navy. These old military posts, no longer needed for fighting the Indians, are now attaining a higher usefulness in the war against consumption.

These various sanatoria, whether private, state, or national, are almost wholly devoted to incipient cases. For advanced cases, the provision, thus far, is mostly limited to large cities, and is made by the municipalities or by charitable and religious societies. Provision is also made in some states for the care of tuberculous insane and prisoners. The segregation of consumptives in the United States, however, is still in its infancy, and is very far from attaining that general and systematic character which alone can have any radical effect in suppressing the disease.

In Europe the conditions are much the same as here. The tuberculosis campaign on that side of the ocean is being pressed with extraordinary vigor. But it is the sanatoria for incipient cases which, at present, are receiving the most attention. Germany, the fatherland of Brehmer and Koch, leads the world in this respect, having over 100 sanatoria with 10,000 beds. Dr. Locke's recent article in the Boston Medical and Surgical Journal gives a most interesting account of the "Crusade against Tuberculosis in Germany," and explains the part taken in it by the Workingmen's Insurance, the German Red Cross and the German Central Committee for the establishment of sanatoria for diseases of the lungs. But, of all this great number of sanatoria, there are very few for advanced cases. The latest report I can find describes only eight, mostly small, but including one large one at Grosshansdorf, with 200 beds, built by the Workingmen's insurance fund. In several German cities, separate wards for advanced consumptives are provided in general hospitals, and much is also being done for this class through dispensaries and polyclinics; but the need of increased provision for these cases is deeply felt. As von Leube says, almost all of the existing German sanatoria are open only to a small minority of the cases—namely, the early incipient, whom he estimates at about 30,000; while, for the advanced cases, of whom there are supposed to be a million, very scanty accommodations are provided.

In England and elsewhere in Europe, the situation is much the same as in Germany. The care of early cases, by sanatorium treatment, is now chiefly engrossing the attention of the civilized world; while the advanced cases, far greater in number and far more likely to spread the infection, remain in their homes, where they are receiving, it is true, an increasing amount of care through dispensaries and visiting nurses. This sort of care, however, cannot prevent

the transmission of the disease to their families. The danger may be lessened, but in the home it is, in the great majority of cases, practically impossible to avert it.

The next step in the campaign will be to undertake the prevention of consumption by separating the sick from the well. For this, two things are chiefly necessary: first, to provide ample hospital accommodation for advanced cases; and second, to establish some systematic supervision over those who, for any reason, cannot be removed from their homes.

Fortunately, in Massachusetts, both of these wants are in a fair way to be supplied through the new laws, but just enacted—one providing for three new satatoria, and one for state inspectors of health. The Commission recommended that these sanatoria should be inexpensively constructed, at a cost of about $500 a bed; and the appropriation is based upon this modest estimate, which is much less than the usual outlay for such institutions, most of them, including that at Rutland, having cost something more than $1,000 a bed. One of the newest in England, the King Edward VII. Sanatorium at Midhurst, cost a million dollars for a hundred beds, or $10,000 a bed, and is no doubt the most expensive of its kind. Since the construction of such sanatoria on a large scale now seems inevitable, a minimum of cost is a highly important desideratum, and if it can be demonstrated that a sanatorium embodying all the essentials, and not of too inflammable materials, can be built for $500 a bed, the segregation problem will be materially simplified.

As we have seen, these three new sanatoria are likely to be filled up promptly with the overflowings of Rutland,—that is, with moderately advanced cases; but the great majority of consumptives will still be left, many of them unfit to be moved beyond the limits of their own town or city. For these, local hospitals are an urgent necessity, and no question can be more important than how to convince the

public of their necessity and how to bring about their construction. The plan of construction is of minor importance. There will have to be an administration building, sufficient for future, as well as present needs, about which may be grouped cheaply-constructed wards, cottages or shacks, which can be increased in number, as needed. These should, if possible, stand in a plot of ground several acres in extent, and, while pure air and sunshine are essentials, the location should be near enough to a centre of population to ensure water-supply, drainage and fire-protection, as well as to permit frequent communication between the patients and their homes. In small towns a farm house may be utilized, while, in larger towns and cities, special buildings will be constructed or special wards added to existing hospitals, as at Fall River and Lowell.

As regards the size of these hospitals, a good rule might be to provide one bed for each thousand of the population, with the expectation of increasing in the future. At this rate, it is evident that, in the smaller towns, only cottage-hospitals would be required. A city of 100,000 population would have a hospital of 100 beds, costing at $500 a bed, $50,000, and the annual cost of maintenance would be about the same as the cost of construction, or $50,000 a year. In cities and manufacturing towns, which have the largest ratio of consumptives, the hospitals will need to be larger in proportion to the population than in the rural towns.

It is to be confidently expected that philanthropic citizens, when they become convinced of the importance of this measure, will help to lighten the strain upon the public purse by donating land and buildings and contributing to a fund for their maintenance.

If all of these institutions are called sanatoria, it may be easier to induce patients to go to them than if they are called hospitals, though the word "hospital" has a far more

cheerful sound in these days than it had a few decades ago.

What then can we members of The Massachusetts Medical Society do, the coming year, to help on the campaign against tuberculosis?

We have been considering two propositions:—*first*, that we are a united body of missionaries who esteem it a duty and a privilege to aid any good cause whose object is to rescue the human race from the tyranny of disease; and *second*, that we have arrived at an important crisis in the world's history, when a great discovery has shown us the possibility of annihilating tuberculosis. The truth of both of these propositions is so obvious that the only possible conclusion is that we must all, individually and collectively, launch ourselves into this campaign, and determine that each year shall become memorable through the achievement of some notable victory.

Encouraged and stimulated by the brilliant successes of the past year, we can now look forward with new confidence and see more clearly where our duty lies.

The *prevention* of tuberculosis rises at the present time into special prominence, and it has been my endeavor to show that the ideal way to accomplish this is through the segregation of the sick. The new state sanatoria will greatly facilitate this separation, and it will be incumbent upon us to induce our patients to go to them, and to convince them that this is best for themselves and necessary for the protection of their families.

We can also agitate for the erection of additional local hospitals for advanced cases, and do what we can to interest people of wealth and liberality in this method of helping their neighbors. If this sort of missionary work is done only by a few of us, the results will be insignificant; but, if we all take hold and do it together, enthusiastically and aggressively, a general and widespread interest will be awakened, and we will have something to show for it, a year hence.

But let me not be understood as urging activity in this one direction, to the neglect of others. The problem is a complex one, and the campaign in many other directions is well under way. It is necessary to continue the attack, with increased activity, along the whole line. Here are some of the ways in which we can render efficient help.

We need to show our approval of the recent acts of the legislature by doing all we can to make them effective. To this end, we must carefully comply with the law requiring notification of cases of tuberculosis to the Board of Health.

We can, whenever occasion offers, explain the importance of the anti-spitting law.

We can cordially co-operate with the state inspectors of health, who are to be appointed, and give them our support in the work they are to undertake, which is expected to result in a systematic organization of the war against consumption.

Moreover, we can lend our aid to the associations for the relief and control of tuberculosis, we can urge the appointment of visiting nurses, the establishment of day camps and dispensaries, we can take pains to see that the disinfection of houses after the removal or death of consumptives is thoroughly performed, and we can bring to the notice of manufacturers the admirable work which is being done in the factories of Providence, of Oxford and of Brockton.

Again, we can accomplish something in our District Societies, by means of the Committees on Tuberculosis, which were appointed year before last, through the active initiative of Dr. A. T. Cabot, then President of the State Society, and now President of the State Committee on Tuberculosis. One way in which the work of these committees can be facilitated, is by providing them with funds for carrying on a campaign of education.

Having done all these things in the capacity of medical missionaries, there is one other thing, which, as medical

practitioners, we must not leave undone. We must seek out the incipient cases and send them to Rutland, or some other sanatorium, while they are yet in the curable stage. We can do this by being always on the watch for danger-signals, by making a careful physical examination in all cases of suspicious cough, of emaciation, of slight rise of temperature, of unexplained debility, by making early examinations of sputum, by not delaying to tell our patients of their condition, and by carefully explaining to them how much better are the results of sanatorium treatment in an early, than in a later stage. We must preach the gospel of outdoor life, and must insist that any of our patients who cannot go to a sanatorium shall have the open air treatment at home.

And even this is not all. We need to keep constantly before the minds of the people that consumption can also be combatted by improving the stock, by bettering the conditions of the working-people, by seeing that they are not deprived of light and air in their homes and the places where they work, and by preventing poverty, vice and intemperance. If those influences can be overcome which tend to sap the vitality of the race, infection will be less frequent even though exposure continues.

Thus the problem of tuberculosis is found to involve the whole science of social economics, and to demand for its solution the combined efforts of all those who are striving in any way to improve the conditions of the human race. This is a holy war, and in it let us, Fellows of The Massachusetts Medical Society, do our full share of the fighting. Let us inscribe on our banners "Consumption not only can be cured, but it can be and shall be *prevented*."

ARTICLE XX.

THE SHATTUCK LECTURE.

THE PAST, PRESENT AND FUTURE OF TUBERCULOSIS.

By FREDERICK C. SHATTUCK, M.D.
OF BOSTON.

DELIVERED JUNE 11, 1907.

THE PAST, PRESENT AND FUTURE OF TUBERCULOSIS.

THERE are obvious reasons why the honor of being asked to prepare the Shattuck Lecture is peculiarly grateful to me. The terms of the will under which the Society has established that Lectureship provide that the income of the bequest shall be applied to the "collection and publication annually of historical or other essays on the climate of said Commonwealth, on the diseases of its inhabitants and on such other subjects as the said Society or its government may select."

It is just one hundred years since Dr. Shattuck, having taken the degree of M.D. at the University of Pennsylvania, at that time the chief medical school of the country, settled in Boston. His father, Dr. Benjamin Shattuck, was a country practitioner in Templeton, Worcester County, an A.B. of Harvard in 1765, but apparently had no degree of M.D., there being no institution in the country at that time capable of granting it, and his circumstances not permitting him to cross the water to secure it.

His life was arduous and was brought to an end at fifty-three, I believe by tuberculosis. This disease also proved fatal to Dr. George C. Shattuck's first wife, probably to one or more young children, certainly to a daughter of twelve. Of seven children, the eldest alone grew up and married. Families of old New England stock are smaller to-day than formerly; but infant mortality is far less among

them, so that the actual decrease in this stock is probably not as great as the smaller number of children would seem to indicate. However this may be, certain it is that the rigorous, changeable climate of New England has long had an evil reputation as being favorable to tubercular disease. This fact, in connection with the loss which consumption inflicted upon Dr. Shattuck's family, his desire to do what he could to make his beloved State healthier and happier, and the prominence which the prevention and cure of tuberculosis has assumed of late years in this community, as well as throughout the world, determined me to choose as my subject for the evening, "The Past, Present and Future of Tuberculosis." The subject may be well worn. It is, however, of vital interest, and I do not feel that I shall wear it out whatever the effect of my remarks may be upon you.

Tuberculosis is no modern disease, its most frequent form and localization in the lungs—consumption—having been recognized and dreaded alike by the profession and the laity in the remotest times of which we have written record. And yet so universal and constant has been its presence in settled and civilized communities, so steady, sure and apparently unavoidable its ravages, that familiarity, although it did not breed contempt, did breed a certain indifference. Until the most recent times it was regarded as one of the manifestations of the inscrutable will of God, almost inevitably fatal,—a part of the necessary order of nature.

Kismet was on the lips of Christian, Jew and Mohammedan alike with regard to consumption. Thus, although dreaded, it was—and is—far less so than those infections which visit us only from time to time and to whose presence and effects we have thus not become so accustomed.

Hippocrates defined consumption as the disease the most difficult to treat and that which proves fatal to the greatest number.

Isocrates, about 400 B. C., is said to have been the first to write on the transmissibility of consumption by contagion.

Aretæus, about 250 B. C., recommended sea voyages for consumption.

Two hundred years later Aritæus, the Cappadocian, thus described the appearance of the consumptive: "voice hoarse, neck slightly bent, nose sharp and tender; cheeks lean, prominent and red; eyes hollow, brilliant and glittering; swollen, pale or livid in the countenance, the slender part of the jaws resting on the teeth as if in smiling, otherwise a cadaverous aspect."

Celsus, early in the first century A. D., advised change of air and outdoor life for phthisis. Let us note in passing that Celsus was a layman who set forth the medical knowledge of his time and of whose encyclopædic writings those on medicine have alone come down to us.

Galen held that phthisis is an infectious process; that there is danger in living with those affected by it. Then came the long, dark night of the Middle Ages when learning, so far from advancing in Europe, fell into a decline, barely kept alive by the monks until the Renaissance. Even the monks, however, devoted themselves to the Latins rather than the Greeks, and were interested in theology and religion, scarcely at all in natural science.

The Arabs, however, revered and tended the lamp of science lighted by the Greeks, and passed it on to the Moors of Spain, and the influx of Byzantine scholars into Italy after the fall of Constantinople greatly stimulated the awakening interest in Greek thought in that peninsula whence it spread northward.

Avicenna, the Arabian, in the first half of the eleventh century, believed consumption to be one of the diseases which spreads from man to man.

Six hundred years later, Fabricius Acquapendente writes, "Celsus amongst the Latins, Paulus Ægineta amongst the

Greeks and Albucasis amongst the Arabs form a triumvirate to whom I confess that I am under the greatest obligation."

Montani in 1550 called consumption one of the most contagious and easily contracted of diseases. With the Renaissance came dissection illuminating anatomy and pathology.

Silenius first demonstrated the association between tubercles or nodules in the lungs and phthisis.

Morgagni would not perform an autopsy on one dead of tuberculosis, and was influential in having regulations passed by the municipal authorities to control the disease.

Antonio Cocci in the first half of the eighteenth century advised the use of sputum cups, and urged the whitewashing of all close, small rooms in which a consumptive had lived. If the room was large and sunny less rigorous measures were deemed necessary.

Lazarus Riverius dwelt on the contagiousness of phthisis and notes its spread throughout an entire family by heredity or contact.

Michel Peter, writing about 1600, says, "I know of nothing more hideously fetid than the bedroom of a rich consumptive. It is a spot carefully enclosed where both air and hope are alike forbidden to enter. There are sandbags to doors, sandbags to windows, thick curtains envelope the bed where the unfortunate consumptive swelters in perspiration, and an atmosphere twenty times respired, twenty times contaminated by contact with his own diseased lungs."

The Dauphin of France, only son of Louis XV. and father of Louis XVI., died in 1765 of consumption. About a year later his wife died of undoubted consumption as shown by the autopsy, probably contracted through her devotion to him. About two months before her death Tronchin, the favorite pupil of Boerhaave, the physician and friend of Voltaire, a man who applied common sense to

medicine, relying on hygiene rather than on bleeding, purging and puking, was called in. In the palace at Versailles it was the custom to close as tightly as possible all windows from November first (All Saints' Day) to Easter. Tronchin found the air of the room so foul that he ordered the windows opened, exclaiming, "The Princess is poisoned." Malice and jealousy perverted the meaning of this remark, which won him he implacable hatred of the Duc de Choiseul. Tronchin was put in charge of the case, fed her generously, insisted on fresh air, made her walk and drive. At first, she improved, the fever abated, and she slept without drugs; but the disease was too far advanced, lighted up afresh, and she did not long survive a large hæmoptysis. A storm of abuse was heaped upon him by the profession. He was called the "worst of charlatans," and was accused of cutting short the life of his august patient by a treatment "as fantastic as murderous." Tronchin is not as well known as he deserves to be, having published nothing on medicine except a monograph on lead colic, which might have been better done. A memoir of him by a descendant was published in 1906 and should be widely read.

In 1760 a hospital was erected at Olivuzza for the accommodation of phthisical patients. In much more recent times the Italians especially, their country being sought by well-to-do consumptives from Great Britain and America, were accused by us of superstitious cruelty towards these patients, the friends of one who there succumbed to his disease being put to great inconvenience and expense during the last illness and the freshness of their grief.

In 1803 Chateaubriand wrote from Rome to a friend concerning the death from consumption of Madame de Beaumont: "I am in great difficulty. I had hoped to get 2000 crowns for my carriages, but phthisis is declared in Rome a contagious disease, and as Madame de Beaumont drove two or three times in my carriages, nobody is willing to buy them."

The great Laennec himself succumbed to the disease, the recognition and pathology of which he had done so much to further. There seems no doubt that Goethe also had consumption and finally died of it.

It may be broadly stated that until within fifty years both the public and the medical profession regarded phthisis as incurable. Efforts were practically confined toward making the patient comfortable and prolonging his life, in a small way toward preventing the spread of the disease, seldom toward curing it.

Early in the nineteenth century, a French doctor, more hopeful than the rest, wrote, "there are two kinds of consumption: that of the rich which is sometimes, and that of the poor which is never, cured."

It is an interesting fact that the two men who stood at the head of medicine and surgery in this community during my student and early professional life had both been tubercular as young men; but both lived to the due age of the Psalmist, and both died of other diseases. I was privileged to hear from the lips of the late Dr. Edward H. Clarke an account of a massive pulmonary hæmorrhage he had between Bagdad and Damascus while travelling for the benefit of his health.

Very great importance was attached to hereditary predisposition, and, diagnosis being less prompt and treatment less rational, the outlook for recovery was indeed gloomy.

I well remember in 1874 hearing a French physician, with whom I was making a visit at the Hotel Dieu at Lyons, remark, "Messieurs, pour la phthisie il n'y a que deux remédes, l'opium et la menterie."

I also heard the late Dr. H. I. Bowditch say that he valued his service at the City Hospital especially for the reason that it helped and strengthened him to see patients get well under his care. You remember that he was the first and leading specialist of his time in Boston in thoracic disease.

Those who could afford to leave home went to Italy, Madiera, Egypt, the Riviera. On this side of the water, Havana, Savannah, Charleston were the main resorts. Dr. John T. Metcalfe during the Civil War gave Nassau vogue, and later started Thomasville, Georgia. He was not a consumptive as have been so many physicians identified with this or that place which they found helpful to themselves and made so to others.

Did time permit it would be interesting to dwell somewhat on the change of view with regard to the essential element of climate for consumption. Warmth and equability to minimize the risk of taking cold were in the ascendant up to about 1860. Then the equable though cold winter climate of Minnesota came into some vogue. Then equability fell from grace, and altitude combined with dryness and sunshine took the first place, especially for incipient cases. We now realize that climatic change, though in some cases very desirable, is not a *sine quâ non* of cure.

Bowditch and Buchanan simultaneously and independently called attention to the relation of soil moisture to the prevalence of consumption, a relation now not much credited.

Some individuals were saved; some had their lives prolonged, yet the general hopelessness of the disease was admitted though the post mortem evidence of puckered and scarred apices had long been known but had failed to teach its full lesson.

It seems to me that ignorance of the pathology of tuberculosis was largely responsible for failure to advance at the clinical end. Laennec upheld the unity of tuberculosis; but the microscope led men astray at first, inducing them to confuse distinctions with differences. We had the desquamative pneumonia of Buhl, the catarrhal pneumonia of Niemeyer, and the dictum of the latter that the greatest danger of the catarrhal pneumonic, that is, consumptive, is

that he become tubercular. We thus wandered in a maze of theory, based on observation it is true, but still theory, for years after Villemin in 1865 proved that the advocates of the infectiousness of tuberculosis were right, and made a step toward establishing the true criterion by which tuberculosis is to be recognized. For clinching knowledge as to this criterion we had to wait then nearly twenty years for Robert Koch and his masterly proof that a special bacillus is the sole cause of tuberculosis, and the indispensable factor in the production of all the multiform changes we had classed as consumption, lupus, scrofula, white swelling, hip and spine disease, etc.

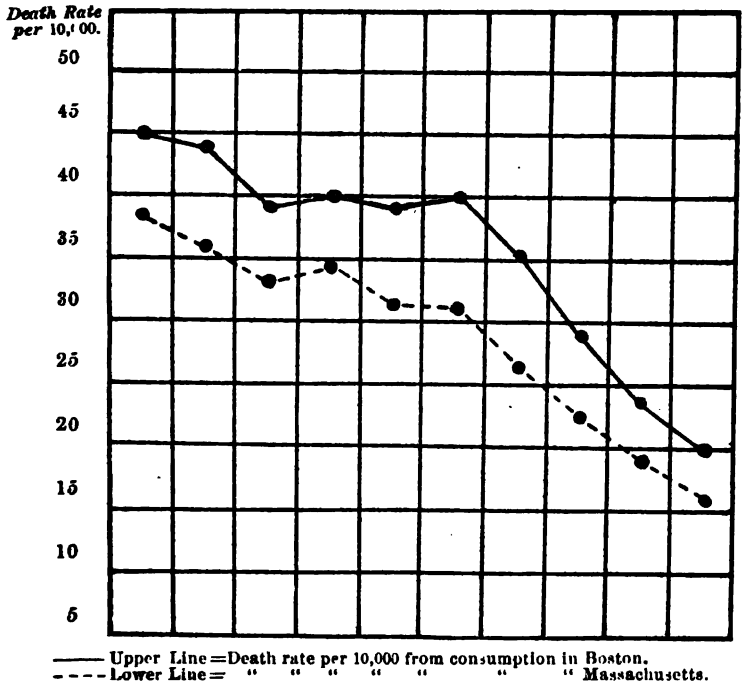
The year 1882 marked a great era in medicine and may be regarded as dividing the past from the present of tuberculosis. We then for the first time began to see clearly, having found the cause, what we had to combat, and could begin to plan the intelligent campaign in which the civilized world is now engaged. The individual who was willing, bravely and cheerfully, to follow the directions of a wise physician and could also afford to do so, had a good chance fifty years ago, though in two respects particularly his chance has improved. His disease is recognized earlier,—a great thing in itself,—and his facilities for securing the prime *desiderata* for recovery are more varied and better.

A sharp contrast between the past and the present of tuberculosis lies in that good hope for recovery is extended from the exceptional to the average individual; in that we recognize and can minimize the danger of the spread of infection; in that, knowing the cause of the disease and its mode of infection, by intelligent and concerted action we can prevent as well as cure. We knew that general hygiene was of the utmost value in the prevention and cure of tuberculosis just as we knew that stagnant water and defective drainage stood in some causative relation to malaria. The discovery that an infected mosquito is the necessary link in

the chain of causation of malaria enables us to act intelligently and efficiently, instead of gropingly and inefficiently, along preventive lines, our cure of the disease in the infected man being practically no better than it was before Laveran discovered the plasmodium malariae.

Let us not forget that a tent on the roof, a shed open to the sun and fresh air in the backyard of a city house, camp-life in the woods, are not "all that there is of the most modern," as the French say.

CHART SHOWING DECREASE IN DEATHS FROM CONSUMPTION IN MASSACHUSETTS AND IN BOSTON.



As appears clearly in charted form, mortality from consumption was dropping gradually in this State from '55 to

'85, since when the drop has been more steady and somewhat more rapid. It is interesting to compare the mortality in Boston with that in the State. From '55 to '65 it fell from 45 to 40 per ten thousand; then was stationary for twenty years, and since '85 has steadily dropped from 40 to 20. The constant influx of immigrants, many of them vulnerable, and their subjection to the adverse influence of urban tenement-house life and its adjuncts may, perhaps, account for the failure of the death rate to decrease in Boston between '65 and '85, while the more rapid fall in mortality since 1885 may well be due to the measures of prevention and cure which are more apt to start in the larger centers.

It is safe to say that formerly scarcely a prominent and wealthy Boston family was free from its case or cases of consumption, often called "decline."

A century ago, and for many years after, women were subjected to many disadvantages. Robust health was hardly ladylike. Fashion in custom and in dress kept them indoors far too much. Outdoor exercise was limited to horseback riding for a very few, a drive or a social call at short distance on foot in good weather. Athletics would not have been tolerated for women even if they had existed. Female footwear was not adapted to the street, still less to country roads, and I have it on good authority that drawers were not worn by women until less than one hundred years ago, it being considered indelicate to clothe that part of the person so nearly after the manner of men. One of the numerous daughters of a wealthy merchant, all of whom long held out against the introduction of drawers for women, was remonstrated with by one of her friends who remarked, "But suppose you should fall down in the street," to which she replied, "A *lady* does not fall down in the street." It is not in flippancy of spirit that I mention these facts. They seem to me to throw an interesting side-

light on the times, and to give a hint as to why practically none of the descendants of those families die of consumption at present. Now and then one gets the disease, but it is promptly recognized and almost invariably cured. It is, however, time to leave the past and to let the dead bury their dead.

Strengthened by and relying on modern knowledge, we are now engaged (I), in the prevention of tuberculosis in all its forms, and (II), the cure and care of those now infected by it.

I. As already stated, it is in prophylaxis especially that we have gained by Koch's discovery. A few words must therefore be said on the mode of infection. The seed is the bacillus of tuberculosis; it is virtually omnipresent in civilized and settled communities; it succumbs readily to sunshine. How long it may retain its vitality in houses, fabrics, etc., we do not know, perhaps not as long as is often and by many believed. Theobald Smith tells me he cannot keep a culture alive more than three months even under conditions most favorable to the longevity of the organism. To his published work and ready kindness in imparting his knowledge I may here express my indebtedness. We are all his debtors. The bacilli of human and bovine tuberculosis are separate organisms today even if they may have had long ago a common ancestor. The bovine form occurs practically only in young children, is rare in them, and the risk of its communication from cattle to man is very small unless the udder is tubercular. Under no other condition can a sufficient number of bacilli gain entrance to the milk to prove harmful. The meat is safe as food. It is therefore irrational, unlimited funds not being at our disposal, to kill all cattle, otherwise well, which react to tuberculin. Much less money intelligently spent in dairy inspection, enforcing cleanliness, detecting and eliminating emaciated and sick cows, will serve all necessary prophylactic purposes.

Tuberculosis itself is very rarely congenital, so rarely that for practical purposes we can leave this mode of infection out of account. Whether the seed of adolescent and adult tuberculosis is planted in childhood, reaching the blood stream and lungs *via* the tonsils and intestines, as is held by so many of the French and by Behring, or not, is somewhat apart from the broadest view of prevention. In so far as they believe the implantation to come from milk they are probably wrong, the non-identity of human and bovine tubercle having been apparently proved. All floors are not clean. Babies in crawling and walking are close to them, and like dogs they are not over-particular as to what they put in their mouths. The seed lives in man and must be able to get from one man to another, to change its dwelling, in order to persist. Its vehicle is the sputum in the vast majority of cases. It is therefore by efforts tending to prevent the preservation and diffusion of the sputum of those affected that we can most simply and efficiently limit the supply of seed, which furnishes such a rich harvest to the Great Reaper. It is fortunate that a remedy which must be generally applied is so simple,—really within the reach of the lowest intelligence and the most slender purse. The community as a whole is rapidly learning its interest in the care of sputum. The individual is, probably less rapidly, awakening to his duty, first, as a member of a family; second, of the community. We must continue to diffuse this knowledge and to enforce as fast and far as we can action in accordance with it.

The only way to absolutely eradicate tuberculosis lies in total destruction of the seed, a conceivable rather than an attainable possibility. It is notoriously a seed which flourishes on poor soil. Whether poor from the start—congenitally poor—, run out in one or more of a thousand ways, or both together, matters little for practical purposes, save that the children of families with a strong tuberculous taint

must be tended with extra care and wisdom. Therefore an important part of prophylaxis is to so enrich the soil, that is to so increase the resistance of every member of the community, that such seeds as may gain lodgement will die without issue, or, at all events, without extension. Thus everything which tends to improve public health is helpful —cleanliness; good and sufficient food, well prepared; pure and ample water supplies; public parks and baths; children's playgrounds; legal restraints upon the greed and stimulation to the inertia which neglects the hygiene of tenements, workshops and factories; cultivation of a taste and provision for the means of outdoor exercise and recreation. These are far from being all, though they are among the most important agencies uplifting the public health. It is to them that the decreasing mortality from consumption up to 1885 is to be exclusively credited. Nothing like an accurate apportionment can be made of the relative value of seed destruction and soil enrichment since that date. Seed destruction is a new factor within twenty years; but during this same period we have seen ever-increasing attention to hygiene in all its branches, especially on the part of the State and the municipalities. The golf course and tennis courts in Franklin Park, the ocean pier at South Boston, public baths, the medical inspection of school children—imperfect as it is as present—these are straws which show the way the wind blows. Straws they are too in another sense, threatening to break the back of that patient camel, the city taxpayer, burdened with an extravagance—or worse —of expenditure, which no private business could or would tolerate. Quick transit tends to diminish the density of urban population. Corporations, partly under the pressure of public opinion and legal enactment, but also from the mingled motives of justice and self-interest, improve the conditions of their employees. Public-spirited and benevolent citizens are forming new organizations, and are also

as individuals helping along the work. All indications point to great vitality in these influences making for the public weal. Individualism, the saving grace of fifty years ago, has been weighed in the balance and found wanting. Its antithesis, socialism, is advocated by some and feared by others, though the soil of the United States is probably less fitted for it than any other on the globe. The legal maxim, *caveat emptor*, is confronted by pure food and other laws limiting the freedom of the individual for the greater good of the community. The individual must be ready to forego privileges, some of them perhaps innocent as far as he alone is concerned. President Eliot well said in a recent address that it is in co-operation that we can attain the best results, and nowhere will co-operation enriched with public spirit yield a richer harvest than in, wisely and along broad lines, striving to prevent tuberculosis.

Among prophylactic measures, those of which Grancher may be regarded as the parent in France must be mentioned. In that country the belief is widely held by physicians that tuberculosis is usually contracted in infancy and childhood, even if it does not develop until adult life. Grancher and his wife started the movement to remove the children of tuberculous parents with their consent to country houses or sanatoria until the children are strong enough to resist the disease. It is claimed that fifty per cent. of those children are cured. There are only two such sanatoria on our greatly extended seacoast. Children with enlarged glands—tubercular—and those debilitated from any cause,—pre- or potentially tubercular—are followed up to their homes and either removed to "Ecoles de plein air" or built up at home by generous diet and instruction of the parents in general hygiene. It has been found far better to enlist the interest and intelligent co-operation of parents in better feeding at home than to furnish milk and eggs and the like

at school. This work is growing rapidly and is in private hands, not, as is usually the case on the Continent, under governmental control or pay.

The proposed extension of medical inspection of the Boston school children and a detail of nurses to visit and teach the families could render great service along these lines, and may, it is hoped, spread widely and rapidly.

II. We now pass on to a brief consideration of the measures at present under trial for the cure and care of those not only infected but diseased. As Theobald Smith keenly observes, infection and disease are not quite one and the same thing. A person may be infected—harbor the bacilli of tuberculosis perhaps indefinitely—and yet remain perfectly well. He is not diseased, strictly speaking, until the infection spreads locally or generally.

Perhaps clearness may be promoted by the rough separation of the advanced and the early cases. I say rough advisedly, inasmuch as the best means to combat tuberculosis is a new subject, experimental, somewhat chaotic, rapidly developing. The leading principles are fairly clear; but we are to learn how to apply them to existing conditions in a decentralized country composed of forty odd sovereign states, presenting often wide divergencies in climate, industries and other important respects, not only between but also within each other.

Measures desirable in and applicable to Germany, for instance, may be entirely out of place in this country. It is in some regards easier to classify for the future than for the present.

Cure can be expected in very few of the advanced cases. In such we may aim at prolongation of life, promotion of comfort and the safety of others.

In the well-to-do the disease is usually recognized early and pretty well cared for at the present time. Among cleanly people with reasonably spacious, sunny, and airy

quarters, the risk of the spread of infection is very small. This appears clearly from the experience of the older hospitals and sanatoria for consumptives in Germany, England and this country. In such hospitals it is not common for attendants to acquire the disease, though Dr. James Minot tells me that six nurses have developed consumption at the Long Island Hospital since 1896. They all entered the training school with a physician's certificate of good health. He does not know whether or not they were examined soon after their arrival upon the Island. City politics are killing to body and soul alike.

Dr. Nichols, however, writes me that during the fifteen years he has been connected with the State Hospital at Tewksbury, one physician can reasonably, and two officers possibly, be supposed to have contracted the disease there. He also states that he has never had any evidence of the spread of the disease from one patient in the Institution to another; and yet during the first half of this period segregation of consumptives was very incomplete, and those exposed, being paupers, were presumably not up to the average standard of resistance. Those who wish so to do can contrast here Boston and the State.

In the House of the Good Samaritan for over fifty years half the adult inmates have been consumptives, generally advanced cases, and no attempt at segregation was made until removal to the new building two years ago. During this half century among all employees one nurse alone contracted the disease within the walls as far as is known. A good illustration of what can be done even under very unfavorable conditions is afforded by the so-called "lung block" in New York. It is in the most congested and crowded district in the world, the ward containing it averaging four hundred and seventy-eight human beings to the acre. This block alone holds nearly four thousand people, of whom some four hundred are babies. It is closely

packed with huge grimy tenements which are honeycombed with rooms. Light and air have been slowly shut out to make more rooms. Walls, courts and air-shafts are cramped, deep and sunless. The body of the block is packed with Jews, Irish and Italians, with a sprinkling of twelve other nationalities. Owing to the comparative immunity to tuberculosis among the Jews there are relatively few cases of consumption in this block in their race. One house called the "Ink Pot" has front and rear tenements five floors high with a foul, narrow court between. Here live one hundred and forty people of whom twenty-three are babies, and here the plague thrives in darkness and filth,—filth in the halls, over walls and floors, in sinks and closets. On the ground floor in one corner where there is apparently little sun and light lives a German family who have kept their windows opened and their rooms well-aired and spotlessly clean. They alone have escaped the disease. Practically every other tenement in the block has had its case of consumption.

It is of the utmost practical importance that the public should clearly understand that while tuberculosis is infectious its infectivity is slight, provided that simple precautions are taken.

Advanced cases, among those who, though not well-to-do, can still provide for themselves reasonably well and therefore usually stay at home, are a very important class to find, educate and follow up to see that they and those with whom they live practice what they are taught. Advanced cases among the very poor, who require so much personal attention, to say nothing of such food and hygiene, as their families and friends cannot provide, are best cared for in hospitals, public and private.

At the State Hospital at Tewksbury, Dr. Nichols writes me under date of March 25th, there are two hundred and forty-seven cases of tuberculosis. In a special building

and neighboring camps and shacks, half a mile from the main hospital, one hundred and sixty men are cared for; but the cases have been sent in such numbers this winter that it has been necessary to put about thirty men in the earlier stages of the disease in the regular hospital wards. Dr. Nichols is asking the Legislature for an addition for fifty patients to this building, merely to meet present and urgent demands. A building for female consumptives, also more than half a mile from the main buildings, will be ready for patients this spring. This is the only public State provision at the present time in Massachusetts confessedly for advanced cases; and to enhance its usefulness, the Legislature a year ago so changed the law as to allow its benefits to consumptives without thus making them legal paupers. The Boston Hospital for advanced cases will be opened before long. There are a few private institutions solely for advanced consumption. There are others which receive such cases in common with other more or less incurable chronic diseases. The present provision is quite inadequate. It is true that less than one-third of the cases at the Rutland Hospital belong to the incipient class. Although it was for that class that the Hospital was designed, not enough incipient cases apply to fill the beds, and more advanced cases are therefore taken. For this state of affairs the Resident Physician blames the medical profession in that it too often fails to make an early diagnosis. This blame is probably in the main deserved, though we all see patients who seek no medical advice until they have passed well beyond the incipient stage. The number of these cases will doubtless steadily diminish in the near future.

The present provision for less advanced and early cases in this State may be classed under:

1. Sanatoria.
2. Dispensaries.
3. Day camps.
4. Tuberculosis classes.

The sanatorium treatment dates back to 1854, Brehmer of Goebersdorf succeeding where Bodington had failed. The latter should be accorded the tardy credit which he did not receive during his life. In 1840, he, "an obscure country practitioner of Warwickshire," published an essay on "The Cure of Pulmonary Consumption on Principles Natural, Rational and Successful," based on the case of a young girl whose faith in him prevailed against the most strenuous opposition of her people and who passed her days, even in winter, out of doors on donkeyback. Although at first her cough was worse, she persevered, recovered, and lived to be eighty-one years of age. He built an institution within which to carry out his ideas, but could not hold out against universal opposition and transformed it into an insane asylum where he later died—a tragic story. Time does not permit even a brief history of the sanatorium movement, but I cannot forbear to mention Dettweiler, Trudeau and Vincent Bowditch. The first sanatoria were private, and our State took the lead in public provision for early cases at Rutland. It will not be many years before every State has one or more such centres, and the movement is now spreading to cities and town.

It is natural that provision should be first made for early, presumably curable, cases. "What can't be cured, must be endured." We like to see prompt results. The educative value of sanatoria for incipient cases is at present great. Every inmate learns what the effective weapons are for fighting his disease and how to use them. After his discharge this knowledge filters, more or less clearly, through him to others who stay at home, and also teaches him, and through him others, how to live without risk to the well. We already see, however, indications of a spreading belief that the greater need is for hospitals to receive advanced cases with disabling and incurable disease, and whose circumstances are such that they cannot at home

receive the care which ordinary humanity demands, or be safe companions for the healthy. The State Commission on Tuberculosis has just recommended the Legislature to establish three hospitals in different parts of the Commonwealth for such cases. It does not advise any more provision for early cases. Institution life at best has serious disadvantages, particularly for those but partially or slightly disabled. Such degree of productive or useful occupation as strength permits is conducive to health. This is difficult to secure in connection with sanatorium life, and it has been suggested that some kind of farm work be an adjunct to Rutland for those who are fit. This can only be carried out during a limited part of the year. An objection common to health resorts and sanatoria alike, whether for the rich or poor, lies in the congregation of considerable numbers of persons suffering from the same disease, and in tendencies to a mental state resulting therefrom. The person who stays at such a place longer than his disease and circumstances in a large sense really demand from that moment gets harm physical, mental, or both. Here, as everywhere in medicine, we must individualize as far as we can, and we can often do so farther than seems possible if we will only compel ourselves to the hard work of thinking.

Such luxurious provision as is made in some of the German institutions is neither necessary nor desirable. It is sheer extravagance begotten of too much money, analogous to that of some of our life insurance companies which have been so cussed and discussed of late. Just as we have begun to see that climate is only an indirect means for the treatment of cases of tuberculosis; that there is no part of the world where the disease does not exist if favorable conditions are present or are introduced, great advance is made in home treatment, which must and should be the only treatment for the great majority of cases.

This brings us to the tubercular dispensary of which Philip of Edinboro was the founder in 1887. The Royal Victoria Dispensary was not only the first of its kind but is so complete today as to serve as a model.

The Edinboro organization aims to provide:

(1) A place where poor patients can be examined;
(2) Where bacteriological examinations can be made;
(3) For instruction of patients;
(4) Dispensary for drugs, disinfectants, food, etc.;
(5) Visits in homes of patients by
    (a) doctors;
    (b) nurses;
(6) Means for the selection of early cases for sanatoria and late cases for hospitals;
(7) For the general guidance of patients and their friends, to answer inquiries from all interested persons on every question concerning tuberculosis.

There are four doctors (one is paid $300 a year), a trained nurse, a volunteer Samaritan Committee of ladies who look after school children and families of patients. There is a paid employe on the premises who helps take histories, etc. The dispensary costs $2500 a year. Interest has been awakened in other cities and towns. There is now a sanatorium for early and a hospital for advanced cases. To complete every such organization, there should be colonies—or some means of keeping track of the after life of arrested or cured cases, and of getting them proper employment, etc.

Calmette at Lille in 1900 opened the Emile Roux Anti-tuberculosis Dispensary. His idea has been taken up in Germany especially by Van Pütter and Kayserling, where numerous cities have opened so-called "Fürsorge-stellen," "Care and Advice Stations." Here instruction is given to patients who must live at home; sputum examinations are made, food and medicine are given when necessary, and

visits are made in the homes of the patients themselves. In Boston in 1899 at the Boston Dispensary this idea was adopted. The Phipps institution, opened in Philadelphia in 1903 through the generosity of Henry Phipps and under the guidance of Laurence Flick, does this on a more thorough scale than almost anywhere else. In New York the Department of Health Clinic for the treatment of Communicable Pulmonary Diseases, established in 1903, is the best example of such work under municipal authority.

Recently in Boston the system of tuberculosis classes has been introduced, and thanks to the enthusiasm and devotion of Doctors J. H. Pratt and J. B. Hawes, 2d, and Cleaveland Floyd, is bearing most promising fruit. This gives a large amount of care to a small number of patients, the reverse of what is done in most dispensaries. The "class" is a unit which can be indefinitely increased. Five such classes have been formed in Boston, two in New York, and one each in Cambridge, Providence, Brockton, Lynn and Baltimore.

Philip of Edinboro and Calmette of Lille are the two great names in dispensary work. Calmette's are called Preventoriums. Besides giving milk, eggs, etc., to poor patients, in some instances the rent is paid, it being considered a good investment.

Day camps originated in Berlin—"Walderholungsstätte," founded by Dr. Becher. Insurance companies pay for the food, and railroads give special rates to and from the camps, two cents for eight miles. These camps are for patients too advanced for sanatoria or who for some reason or other must live at home. There are two such in Boston, the only two in America. Berlin has three, the only ones in Europe.

The weapon against tuberculosis is education: education as to how it is spread, how prevented; how treated for cure and alleviation, and for safeguarding the community. The

campaign is on. Earnest, devoted leaders have come forth from the profession, working with small thought of money return. The public is ready to help, to co-operate with brains, time, money, service. Are we doctors as a body doing our full share? I doubt it. Too many of us I fear —I myself cannot enter the plea of not guilty—are more or less afflicted with inertia, congenital or acquired. There is a large supply of enthusiasm everywhere among the laity, which only needs the direction of physicians to bring new land under cultivation as well as to increase the yield of that already planted.

We cannot realize too strongly that everything which tends to elevate the public health, directly or indirectly, makes the struggle for existence of the tubercle bacillus harder. At one or another place in the world almost every necessary specific step for prevention or cure is taken. One place or country has given special attention to one branch of the work and gone far in it; another to another branch. Some places have done little; too many nothing. The work of the immediate future, therefore, would seem to lie in generalizing and systematizing the work in conformity with the varying modes of government and administration of each country and independent state, with due consideration of its customs, conditions, industries, *et cetera*. Modification of details must be made to meet the different demands of large cities, the factory town, the village and rural community. Just as good results can be secured where local self-government prevails as under a centralized and paternal system; but people must be made to see their importance and to desire them, securing proper legislation and ordinances, and providing means for the enforcement of the same. In our own State, the work for the immediate future would seem to lie in:

1. The passage of a general law making the reporting of consumption compulsory. But no such law can be thor-

oughly efficient unless the medical profession as a whole is competent to make an early diagnosis, and is in such sympathy with the law that the physician puts obedience to it and its underlying principle above the selfish preference of this or that patient and above any fear that he may lose practice by reporting him. It is not likely that such opposition will be very serious or long-lived.

2. Laws providing for the disinfection of dwellings occupied by the consumptive after moving or death.

3. An extension of the anti-spitting law, so as to include at least factories, mills, schools, and the halls of tenement houses.

4. Legislative provision for State Medical Inspectors, responsible to the State Board of Health, each in charge of a district within which he supervises organized work.

5. Each community in the organization should have such force of nurses, visitors, or both as each may need; that the poor may be followed up in their homes, taught what to do, helped to do it, if necessary, and encouraged to persevere in doing it. Much of this can be done without, or with trifling, expense. There is much intelligent energy running to waste which can be turned in this direction.

All the above recommendations are made by the State Commission to investigate measures for the relief of consumption. Their report should be read and pondered by every physician.

Immense service can be rendered by arousing the interest of private and incorporated employers of labor in mills and factories. When the example set by the A. L. Joslin Co. in this State and by the Sharpe & Brown Co. and Mr. Metcalf in Providence at the instigation of Doctors E. P. Joslin and Fulton is generally followed, the mortality returns will tell the story, and there is no reason why it should not be followed if we physicians do our duty. Mill owners and managers are as a class intelligent and humane. Let their

physicians call attention to this matter. It is highly probable that there will be no economic loss to the employer. There is more likely to be a gain to him, and the gain to individual workers, as well as the community, is sure to be great.

The tuberculosis exhibitions which have been held are potent aids in teaching the public the possibilities of prevention and cure. Such exhibitions should be brought within reach of the great majority of the inhabitants of the State. While the campaign is primarily against tuberculosis, success in its prosecution must indirectly diminish the liability to, or serious effects of, many other diseases more or less intimately dependent on impaired vitality.

Past experience proves that the mortality from tuberculosis can be greatly reduced. There are those who go so far as to say that a systematic and energetic fight will render tuberculosis as rare as leprosy. Unless a vaccine comparable to that for small-pox, or some other means producing artificial immunity, can be discovered and universally applied, the above view would seem too sanguine. The problem of leprosy was a relatively simple one. That disease is far more difficult to transmit from one individual to another than is tuberculosis. Its lesions are mainly external and it is thus more easily recognized. The segregation of leprosy carried out during the Middle Ages could thus yield results hardly to be hoped for in the case of tuberculosis, an adverse factor in the attempt to stamp out which lies in that, unlike most of the more acute infections, it can be carried and perpetuated by a number of domesticated animals—cats, dogs, rabbits. The tubercle bacillus is being put on the defensive and we can be sure will fight for his life just as we are fighting for ours. Is it not possible that more resistant and virulent strains may develop, if not wholly, at all events partly, in proportion as we develop immunity? That nature is very careful of the species

while seemingly regardless of the individual is as true of a bacillus as of larger organisms. Man has totally exterminated the great auk. The Labrador duck has utterly disappeared since 1860 without any assignable cause. The bison and the wild pigeon have practically disappeared. Animals which breed slowly, are defenseless, and like the bison, require vast tracts of land coveted by civilized man, are handicapped in the struggle for existence. I am told that the few herds of bison which are preserved only just about hold their own, showing very slight, if any, tendency to increase. The balance of nature is very complex and may be very delicate. It does not seem probable that the disappearance of the wild pigeon, such enormous flocks of which still existed easily within the memory of many of us, can be attributed save in subordinate part to their slaughter by man. The part played by man in the extinction of the bison is probably larger than in the case of the pigeon. The preserved herds of bison are well cared for but can no longer migrate. Why do not they increase under protection as do moose and the white-tailed deer? Is it not possible that these beings had about reached their limit of adjustment to external conditions and that any slight change in the balance against them was too much for them? Our lives, and even the written records of man, are so short, and it is so hard for us, busy with our daily struggles, to project our thought and imagination far enough backward or forward. Man himself is a mere babe in arms in comparison with life on this planet, and his parasites in their present form must be younger still. The destructive moths which have caused so much trouble in these parts of late years find no adequate natural enemies in this country. In Europe they ordinarily are kept within bounds, but even there they now and then get ahead of their enemies for a time by reason of some change in the balance.

This line of thought may not be practical. It certainly

does not materially influence action now or in the immediate future, but it seems to me deeply interesting and wholly pertinent to our subject of the evening. There is no tendency to race suicide in the tubercle bacillus. He finds an acceptable *habitat* in man, whose numbers are increasing, and whose tendency in civilized countries to form large communities runs directly counter to some of the measures which he takes to protect himself against the bacillus. We know that we are decreasing and can further diminish the mortality of tuberculosis. Can we correspondingly decrease the morbidity? I doubt it. Tuberculosis seems likely to persist for a long time to come, perhaps indefinitely, in latent, more superficial, milder and more chronic forms. There can be no question that there are many persons—in all probability there are some such in this very room—who have or have had tuberculosis, even of the lungs, and who tolerate or have recovered from it without knowing that they have, or have had it. Here again comes in the distinction of Theobald Smith between infection and disease. It is mainly to his suggestion that I owe the above line of thought.

If the master-word in medicine is work; that in the tuberculosis fight is education. Every teacher knows this to involve work—hard work. To paraphrase Talleyrand's definition of the main requirement of the diplomatist we can say "de l'éducation, encore de l'éducation, toujours de l'éducation."

The effort to compress so large a subject into reasonable limits has not been an altogether easy one. I trust that my sins of omission and commission may not be as obvious to you as they are to me.

ARTICLE XXI.

THE CHANNELS OF INFECTION IN TUBERCULOSIS, TOGETHER WITH SOME REMARKS ON THE OUTLOOK CONCERNING A SPECIFIC THERAPY.

By THEOBALD SMITH, M.D.
OF BOSTON.

READ JUNE 11, 1907.

THE CHANNELS OF INFECTION IN TUBERCULOSIS, TOGETHER WITH SOME REMARKS ON THE OUTLOOK CONCERNING A SPECIFIC THERAPY.

In choosing this somewhat well-worn subject in answer to an invitation to speak on some aspect of the problem of tuberculosis, I was led by the present rather unusual activity in the study of the digestive tract as a portal of entry. This bustling activity is due in the first place to the somewhat morbid tendency of research to seek out the new, rare and unusual, and magnify and illuminate them so that to our dazzled eyes all the laboriously reared truths of the past become invisible for the time being. It is furthermore due to the dicta of two prominent men, one of whom proclaimed that bovine tuberculosis was of no consequence as a source of the human disease, and the other that all tuberculosis originates in infancy, and that much of it was due to cow's milk.

The widespread movements tending towards the education of the public concerning the nature of tuberculosis and the modes of diffusion and transmission of the bacillus, had brought to the foreground the older theories that tuberculosis is largely an inhalation disease and that the chief source of infection is the coughing consumptive. While all agree that the sputum is the vehicle in which the bacilli leave the body, that there is, in fact, no other mode of excretion of the bacilli in man which can be seriously entertained, there has developed a marked disagreement, or

at least an attitude of assumed ignorance, as to the chief portal of entry. While inhalation has been and is still looked upon as the most frequently used mode of entry by some, others have gone so far as to deny the aërogenic infection of the lungs completely, and they trace pulmonary disease through the avenues of the intestinal mucosa and the associated lymph nodes, or the tonsils and cervical lymph nodes. In other words, the tendency on the part of a certain group headed by von Behring has been to cut out entirely the direct introduction of tubercle bacilli into the lungs through the respiratory tract and replace it with certain more circuitous routes, dealing with ingestion rather than inhalation.

Associated with and largely responsible for this change of view as to the significance of ingestion tuberculosis is cow's milk. Were it not for cow's milk as an occasional carrier of bovine tubercle bacilli we should probably have heard little of ingestion tuberculosis.

INGESTION AND INHALATION TUBERCULOSIS.

We have thus two main questions before us. 1. Is the path of the tubercle bacillus to the lungs chiefly by way of the digestive tract? 2. To what extent is bovine tuberculosis responsible for the human disease?

The difficulty encountered in taking a definite position on the question of the paths of the tubercle bacillus both without and within the body is due to the fact that almost anything we may say concerning the tubercle bacillus has a grain of truth in it, and may be realized at one time or another by observation and experiment. The goal towards which we should strive, however, is to discover the ordinary, common paths and not the merely possible or extraordinary ones. Even in the experimental disease produced by tubercle bacilli in the lower animals we occasionally encounter peculiar localizations. The cryptogenetic tuberculosis of

the bones, the kidneys and other organs wholly outside of the usual paths of the tubercle bacillus in the body, shows well the occasionally irresponsible activities of this organism.

The character of the disease started by the tubercle bacillus, like that of other infectious diseases is dependent on a number of variable factors. Among the most important are: — the virulence of the infecting organism, the age of the patient, the inherited susceptibility or predisposition, a previous infection, accidents which convert a latent or healing local, into a generalized disease, and finally the mode of entry.

As regards the virulence of the infecting organism, I must admit that our methods are not perfect enough to disclose minor differences. Until the facilities of the average laboratory extend to the breeding and rearing of our experimental animals, our tests upon animals can only be relied upon to distinguish such gross differences in virulence as exist between the bovine and the human type. There are, however, differences between cultures of the human type which impress themselves upon the investigator as more or less constant, although it would be difficult to define them readily. We may safely assume that a considerable number of slightly varying races of human tubercle bacilli exists, and it is probably not a matter of indifference to the patient which of them is at work.

Age is a most important factor, as all statistics and experimental enquiries show. In this respect tubercle bacilli do not differ materially from other infectious agents which find their easiest prey among the young. Statistics prove that tuberculosis becomes rapidly generalized in infancy and that it is a highly fatal disease. The conception of latency which has been introduced into the subject in recent years by von Behring does not at all tally with such statistical and pathological researches. That a bacillus which is specially virulent for the young should lie fallow

in some organ or tissue of the body without multiplying is, to say the least, revolutionary to our current conceptions. Experimenters have been for years familiar with this conception of latency, or persistence, of infectious agents in the body of those who are relatively immune, and of those who have recovered from a disease, but not with the idea that bacilli can be latent in the highly susceptible young. It is possible that such bacilli may be very feebly infectious, and that subsequent disease is not due to them, but to later infection with more virulent types. It is furthermore possible that certain antibodies may be transmitted from mother to child, and that these are slowly excreted and disappear after three or four months unless the mother's milk continues to supply them. Under such conditions it is conceivable that in the earliest months of life, or even longer, during the period of lactation, bacilli may be restrained and then multiply subsequently.* In any case I doubt that such latency could be prolonged over a year at the longest. The subject is a difficult one at best. How we can tell in any case that there has been no fresh infection where we suppose the disease to have been latent, I do not see. The possibility of fresh infection with perhaps more virulent material must always be reckoned with.

The influence of inherited predisposition though an unknown quantity, cannot be given up at present, and I believe it plays an important part in the fight with tuberculosis. The capacity for producing antibodies, for phagocytosis and the other protective factors probably varies from individual to individual, and has its definite, prescribed limits.

The influence of one attack of tuberculosis, provided structures have not been seriously damaged, must be considered a distinct asset, since we now know that immunization of cattle with human tubercle bacilli is possible.

* In a former paper I advanced another hypothesis to explain such latency of tubercle bacilli. The Parasitism of the Tubercle Baccillus, etc. Journ. Amer. Med. Assn. 1906.

The influence of accidents, such as trauma, intercurrent diseases, secondary infections in favoring dissemination of the disease, is so well known that I merely mention them.

With all these factors at work and influencing the process, it is obvious that our inferences and conclusions as regards portals of entry, dissemination, and the results of different kinds of treatment, must be cautiously made, for they cannot safely rest on a few in part faulty experiments, which may strain and largely overpower the physiological protective mechanisms, and which may fail to reproduce the usual conditions present during spontaneous infection, but they must also take into consideration the work of pathological anatomy and histology, and utilize the results of statistics collected at the autopsy table.

Coming to the first controverted question, whether all tuberculosis is chiefly or exclusively alimentary in origin as maintained by some today, we find that two different lines of research are involved in it: —

1. The anatomical and physiological problem of the absorption of tubercle bacilli, soot, etc. by the mucous membrane of the intestines and their conveyance to the lungs and thoracic lymph nodes without being stopped by the mesenteric nodes.

2. The behavior of the organism in the presence of tubercle bacilli in any of its tissues under the influence of different degrees of a specific resistance or immunity.

As regards the passage of bacilli through the mucosa of the intestine it may be said that some twenty years ago the opinion was quite general among pathologists that the tubercle bacillus produces a lesion at the point of entry. With the more exact and detailed study of tuberculosis in infants and children, and the disease in cattle, this position had to be gradually abandoned. To-day most authorities will admit that the tubercle bacillus may pass through mucous membranes without leaving any pathological traces of its passage, to settle down in the nearest lymph nodes.

How the bacilli gain an entrance into the lymph vessels of the mucosa, whether by the aid of normal, physiological processes, or through minute lesions, is not determined. Their next station would be the mesenteric lymph nodes. The original claim held by many today is that in the mesentric lymph nodes the tubercle bacilli come to rest and multiply, producing distinct lesions. If they escape it is only after leaving the well known traces of their passage behind.

Within the past four or five years a further modification of the original position has been adopted by some, namely, that tubercle bacilli may not only pass through mucous membranes, but also through the related lymph nodes and settle down and produce disease at a point quite distant from the point of entry, without leaving any intermediate traces of their passage. It is obvious that if they escape through the mesenteric lymph nodes their next stopping place would be the capillaries of the lungs and as a result we would have a primary tuberculosis of the lungs due to swallowing tubercle bacilli. That this is possible few pathologists will deny, but is it probable and is its occurrence of sufficient frequency and importance to permit the overthrow of the aërogenic infection in phthisis? Two lines of experiments have been made to prove this. Dogs have been fed tubercle bacilli in milk or other fatty foods, and the tubercle bacilli found in the thoracic duct some hours later. Cattle have been fed with tubercle bacilli with stomach tube and slight tuberculosis of the lungs produced. Animals have been fed with various finely divided powders, either with or without stomach tube, and the pigments found in the lungs. These various experiments, though plausible on the surface, do not, in my mind, prove the contention. The large number of bacilli introduced may have overpowered the usual protective agencies, there may have been aspiration into the air tubes following withdrawal of the stomach tube,

and finally other investigators claim that if pigments are fed in the usual way and kept wet they are not found in the lungs subsequently.

Perhaps the most opposition has been aroused by the attempts to trace pulmonary anthracosis to the ingestion, rather than the inhalation of soot, a process firmly estabtablished by the exhaustive experiments of J. Arnold over 20 years ago. Without delaying over the experimental data, let me call attention to the fact that large amounts of fine particles are taken into the digestive tract by our domestic animals daily in their food, and if any absorption takes place it would be detected. The mesenteric and meso-colic lymph nodes of the omnivorous dirt-eating pig are free from pigment. In the ox, the mesenteric lymph nodes of the young are quite free from any pigment, but when the milk diet has been replaced by the usual diet of grass, hay and grain, these nodes become heavily pigmented, not with soot, etc. but with pigment particles derived from the blood. When we turn to the thoracic lymph nodes in the same species draining the lungs, another picture presents itself. At first these nodes are unpigmented, but as the animal grows older a faint subcortical line of pigment shows itself which grows heavier and denser with age. This consists of black particles and translucent fragments. In the horse similar conditions prevail. A delicate black tracery of the subpleural lymphatics in old animals indicates a frequent deposit in the lungs as well.

It is of interest, furthermore, to note that in cows the primary foci of tuberculosis are situated in the thoracic lymph nodes among the pigment, suggesting most convincingly the aërogenic source of both. If this pigment in these nodes comes from the intestines, why does not some of it remain in the mesenteric nodes? Why do these instead retain in large numbers, chiefly in phagocytes, the blood pigment, and why is this not passed on? Possibly it may

be answered that a special chemotactic affinity exists between the phagocytes on the one hand, and the blood pigment on the other, which does not exist between phagocytes and soot, etc. But if this answer is true we surely cannot pass over the still more important chemotactic affinity between tubercle bacilli and the cells of the mesenteric lymph nodes.

A careful reading of the recent literature shows that the promoters of the theory that tubercle bacilli may readily pass from the intestines to the lungs without stopping to multiply, and thereby produce disturbances on the way, have not taken sufficiently into account the fact that tubercle bacilli are not like grains of lampblack, but living organisms which arouse a more or less prompt reaction on the part of the invading tissues, and which cannot travel about the body at will without being recognized and held.

The varying conditions of immunity and predisposition have much to do with the localization of tubercle bacilli, and the shaping of the disease process. It is evident that the different stages in life do not react alike towards tuberculosis.

What may be true in earliest infancy is not absolutely so in childhood. Even puberty, middle life and old age have their respective types of tuberculosis from which it is true there are deviations, owing to the variable factors already mentioned, but there is as a rule a clearly defined growing immunity with age. There is even a varying immunity of the different organs of the same body towards the tubercle bacillus, and this may not be the same at different ages.

The lungs may be said to be the *locus minoris resistentiæ* for tuberculosis not only for man, but for the higher mammals as well. Disease starting elsewhere, if not checked by death, or recovery, sooner or later leads up to the lungs. In my second paper on bovine and human bacilli, published nine years ago, this behavior of tubercle bacilli was fully

appreciated. It is there stated: — "The question of phthisis as secondary to infection by way of the digestive organs is, however, one needing more attention . . . . . In all mammals the lungs are evidently the most favored place of tubercle bacilli, and wherever the latter may be deposited, they sooner or later, unless the disease is checked, reach those organs, where the process spreads more rapidly than elsewhere."

In the attempt to establish the alimentary origin of tuberculosis, some have based their arguments on the fact that inoculation into different parts of the body, even into the tail, may lead to the pulmonary disease. This fact has been known for a long time, and has been mentioned by various observers. The question before us is not whether the lungs can be made diseased secondarily. That this is possible, and occurs frequently, as in miliary tuberculosis, is well known. The real question is whether the usual types of pulmonary tuberculosis appearing at some one point in the lungs start from the intestines without leaving any signs of their passage, or whether they are the result of inhalation. That is to say, is the pathologist wholly powerless to trace the paths of the tubercle bacillus in the body? I think not. With the aid of our knowledge concerning immunity, the following general laws may be laid down: —

In infants, the entry of tubercle bacilli is easier through all portals, and a generalized tuberculosis results from the rapid multiplication of bacilli and their diffusion through the feebly resistant tissues and vessels. With increasing age this indiscriminate passage of bacilli is checked, and the bacilli are held back by the reacting tissues in the nearest lymph nodes, the so-called regional nodes. This gives us the common types of tuberculosis of childhood. It is also the type of the disease in young cattle. With increasing resistance, as at puberty and later, we have certain

portals of entry, such as throat and intestines completely closed under ordinary conditions, and an inhalation disease appearing almost exclusively because the lungs, the most vulnerable organs, are still accessible through the respiratory passages.

COW'S MILK AND TUBERCULOSIS.

Closely interwoven with this new or revived doctrine of the alimentary origin of pulmonary tuberculosis, is the widespread belief or assumption that milk infected with bovine bacilli plays a large part in the etiology of human tuberculosis. The history of this belief is of interest in showing the to-and-fro swing of medical doctrines. Following the epoch-making study of Koch in 1882, the assumed identity of bovine and human tuberculosis, as formulated by him, provided a basis for the world-wide movement against bovine tuberculosis as a source of the human disease. Excessive zeal in this direction was, however, kept in check by the scarcity of alimentary tuberculosis and the universally accepted doctrine that pulmonary phthisis was an inhalation disease.

In 1896 and 1898 I demonstrated the existence of two types of tubercle bacilli, the bovine and the human; emphasized the inadequacy of the evidence on which the transmissibility of bovine tuberculosis to man was founded, and pointed out that with the aid of these two types we could now build up more satisfactory statistics. In 1901 Koch, who had meanwhile studied the same subject, in a now memorable address claimed that owing to the difference between human and bovine bacilli, bovine tuberculosis was a negligible factor in the human disease. As a result of this address, commissions were appointed in different countries to investigate Koch's claims. The German Commission reaffirmed the existence of the bovine and the human type, as representing distinct races of tubercle bacilli. The same

has been done by the English commission in their recent report. But the latter claim that these two types are simply extremes of an intermediate series of transition forms and admit, with considerable reserve however, that there is some evidence of mutation, and that perhaps the bovine type may be converted into the human type by a sufficiently prolonged sojourn in the human body.

Coincident with this study of the relation of bovine and human tubercle bacilli to one another, nearly all observers who have studied series of cases of the human disease have found, in certain cases of tuberculosis, bacilli of the bovine type. The labors of Ravenel, the author, the German and English Commissions, Rabinowitsch, Fibiger and Jensen, and others have collected between 40 and 50 cases of the bovine type in infants and children. Some cases were fatal, others not. In practically all these cases the disease started either in the tonsils and related cervical lymph nodes, or in the mesenteric lymph nodes. So far as I know only one bovine culture has thus far been isolated from sputum, and this by the English Commission. Together with two other types it was isolated from a calf fed with large amounts of mixed sputum.

The cases from which these bovine bacilli were isolated are all selected cases, and we have at present no way of determining what percentage of progressive fatal tuberculosis is due to them. The statistics of alimentary tuberculosis vary from place to place, and even these cases are largely associated with the human bacillus. The only way to gain some adequate insight into the amount of damage done by bovine bacilli would be to take a series of consecutive cases both in sanatoria for adults, and in children's and infants' hospitals, and study the bacilli of every case for a given period. I venture the statement that probably not more than one per cent. of all cases will show bovine bacilli, and that in individuals over 12 years they will be found only

very rarely. However, this is a mere guess and it should not take the place of actual figures.

The methods of isolating and identifying tubercle bacilli of the two types is now so far advanced that the clinical laboratories should take up this study. A series of cases carefully studied clinically as well as bacteriologically may lead to some means of clinical diagnosis between the two types of disease, since the bacteriological diagnosis under ordinary circumstances compels us to wait at least three months before a decision can be made. It is also highly desirable that any aberrant or peculiar type of tuberculosis be studied bacteriologically in order to determine the nature of the invading bacilli.

The issue then would appear to be clear-cut and it would seem as if the whole question might be solved bacteriologically. But those who insist on the old doctrine of indiscriminate transmissibility of the two types of bacilli, and who are fortifying themselves by attempting to establish the doctrine of the general alimentary origin of tuberculosis go a step farther, and assume that the bovine type may gradually change into the human type in the body, so that the adult phthisical patient would discharge the human type in his sputum, whereas he may have absorbed the bovine type earlier in life through the digestive tract. This is perhaps a plainer and franker statement of the hypothesis than its adherents have been willing to make, but there can be no harm in viewing its bald outlines. The possibility of such a transformation I took into account as far back as 1898, and stated it as one of the theses to be investigated, although I did not then, nor do I now believe in its realization.

The only way in which such an hypothesis could be tested would be to find a species of mammal in which the human type is always found, perhaps some race of monkey, and pass the bovine bacillus through a series of individuals. But the possibility of latent foci in such animals might at

any moment imperil the accuracy of the experiment, by bringing into our cultures a human type from outside sources.

That the tubercle bacillus possessess a certain plasticity has been brought out by various investigations. Cultures from domestic animals other than bovines, but evidently derived from the latter, I have found slightly different as regards virulence from parallel bovine cultures. But that the bovine type should be converted into the human type has no analogy in the domain of bacteriology, and as long as it is not clearly proved to occur, it cannot be accepted on mere speculation. Again, if the bovine and the human cultures are indiscriminately transmissible to either species, why should two types have arisen? These must have been developed from some parent stock in obedience to the demands of the respective hosts harboring them.

Bacterial species producing specific invasive diseases in man and animals have their congeries of characters well fixed. Degeneration of such characters may occur, but the transformation of one into another even closely related race or variety has not been accomplished. Nocard claimed to have transformed mammalian into avian tubercle bacilli, but this solitary experiment should be repeated and tested with the aid of our present knowledge of the tubercle bacillus before it can be fully accepted.

The transmission of infectious animal plagues to man occurs in the case of glanders, anthrax, rabies and bubonic plague. In these infections which are due to bacteria, only one type of microbe is known, not two. The transmission of glanders to man occurs rarely when we consider the large number of glanders-infected horses in our midst, and the opportunity for infection. Man is predisposed only to a limited degree, and the transmission of infection requires without doubt certain specially favoring conditions, as seems to be the case in man when exposed to the bovine tubercle bacillus. If, as is claimed by some, the bovine

and human types of tubercle bacilli simply represent a connected series, and if there is no sharp distinction, it is rather strange that adult man should harbor the least virulent type. Usually, the older the attacked individual the more virulent the attacking organism, for it is the old that infect the young.

To look towards another species for the main source of contagion in a human disease which has its own bacillus and machinery of transmission is a bacteriological anomaly, which has no parallel at present, not even in the bubonic plague. The anomaly is in fact so great that no experienced bacteriologist who has studied pathogenic species over any period of time is found to favor it. At the same time, I believe that we should not stop in our investigations, but work out the problem in every direction. If an anomaly is really discovered and proved, we must accept it. But I believe it wholly premature to base upon it far-reaching practical measures, whose cost to the community might destroy the opportunity of saving many times more lives than are injured or destroyed by bovine virus. In short the entire field of bacteriology may be hunted over without avail to find a precedent for the assumption that two distinct races of bacilli adapted to two widely separated hosts, such as the ox and man, are indiscriminately transmissible. All that we can say is that in certain receptive stages in life characterized by a low degree of resistance such transmission may occur, and it does occur under circumstances not yet fully known. I am, therefore, of the opinion that man has only a limited susceptibility to bovine tuberculosis depending on certain still unknown factors. It is from this standpoint that I shall discuss very briefly the practical side of the subject.

The recent attempts to prove all tuberculosis of alimentary origin, if seriously entertained and if finally proved, would lead to a certain readjustment in our sanitary measures.

The tuberculous patient would not only be dangerous near us, but at any distance, provided he came into any relations with us. Those who prepare our foods could infect us at any moment. The bread and pastry we buy, the milk we drink if handled by a tuberculous patient, even the clothes we wear would bring an element of danger. Every act of coughing would be dangerous, even if the sputum were carefully destroyed, because the spray might settle down and infect our food.

It seems to me that before placing an additional ban on the tuberculous we should patiently wait for satisfactory proof, and not be stampeded from one position into another before we have inquired into the qualifications of the innovators, and the accuracy, if not reasonableness, of their statements. The problem of bovine tuberculosis should be dealt with in a similar temperate manner. We have learned during the past nine years that there is a bovine and a human type of the bacillus which all reliable bacteriologists have recognized. We have furthermore learned that in a selected number of cases of alimentary tuberculosis the bovine bacillus has been found. This is all we know and all the rest is uncertain and speculative.

As regards the source of the bacillus, we know that it may come from the udder or from the feces, when sufficient cleanliness is not observed. The udder becomes tuberculous through metastasis in perhaps one per cent. of tuberculous cows, roughly speaking. The parenchyma or secreting structures become the seat of tubercles, and the milk may contain in certain cases large numbers of bacilli. When children are fed with such milk, they are overpowered by the many bacilli, and where a few might pass through, of the large numbers daily ingested, some invade the body. When udder tuberculosis exists in a herd samples of the mixed milk may prove infectious for guinea pigs, because the large numbers shed by one animal are disseminated

through the entire mass, and enough may be present to make all the milk infectious for guinea-pigs by inoculation.

There has been a vigorous discussion whether cows which simply react to tuberculin and which show no clinical signs of tuberculosis do or do not shed bacilli in the milk. Some observers contend that they may do so. Others deny the contention. My own experience coincides with the latter. Ostertag, who made a careful study of this subject for the Prussian government and who found no tubercle bacilli in the milk of reacting animals, pointed out that the discrepancies between observers may perhaps be explained by the soiling of the udder with feces. In cows with a discharging focus in the lungs, the swallowed sputum would maintain a slight infection of the feces. He drew the milk himself, and carefully cleansed the udder. In one case in which the feces were infectious, the udder when left uncleaned yielded milk which produced tuberculosis in guinea-pigs. After due cleansing it failed to do so.

If Ostertag's interpretation is correct, and I am inclined to believe it is, a careful periodical inspection of dairies by competent and conscientious veterinarians followed by the removal of clinically tuberculous animals, and all that had any suspicious abnormality of the udder, and the enforcing of cleanliness would eliminate practically all danger. Some chances for slight infection of the milk may still remain, as is equally true with typhoid fever, diphtheria and other infections in the dairy, but the amount of infection would be greatly reduced if not entirely eliminated and in tuberculosis more than in any other disease, I believe that the number of bacteria plays a very important part in the final infection of the human subject.

THE RESTRICTION OF BOVINE TUBERCULOSIS.

In view of the relatively very small percentage of cases of transmitted bovine tuberculosis, and the very slight

chances for the infection of the milk of cows in the early stages of the disease, the question, what policy designed for the protection of the public will do the greatest good, is a perplexing one.

It seems to me that competent dairy inspection, the removal of all animals which show evidence of udder disease and which from a clinical examination can be pronounced tuberculous, and the enforcement of strict cleanliness are what should be insisted on.

This supervision, as I pointed out over ten years ago, not only greatly reduces the chances of tubercle bacilli in the milk, but it also restricts and suppresses two other factors which injure or destroy probably a hundred times more children than the bovine baccillus. These factors are miscellaneous putrefactive and fecal bacteria from filth, and the germs of specific human diseases accidentally introduced by those concerned in the production and distribution of milk.

About ten years ago the hope was entertained in some quarters that bovine tuberculosis might be completely exterminated by some concerted and persistent effort. While the attempt was in itself laudable, it overlooked certain obvious facts, and it was planned on such a scale that it was bound to fail. It was based on the methods of procedure of the National Government in its fight against bovine pleuro-pneumonia and foot-and-mouth disease. The nature of these two diseases, their relation to other species, the character of the virus and its diffusion over a limited well-defined territory made the task of suppression a reasonably certain one, even though the cost was great. But bovine tuberculosis differs so widely from these infectious diseases in almost every one of the details which have made the others manageable. To suppress bovine tuberculosis it will be necessary to have the coöperation either compulsory or voluntary of those who produce milk, and to estab-

lish, so to speak, islands or centres of tuberculosis-free animals, and slowly increase such territories. To look to our State and National governments for paternalistic aid to control a disease with which the farmers are now well acquainted will not work even if it were just to the rest of the population. The only way to make any progress is for the consumers to insist on milk from inspected herds and pay the price necessary to keep them free from disease.

In our own state, the Board of Health inspects dairy herds and the Cattle Bureau removes, pays for and slaughters, clinically diseased animals. It also tests all animals coming in from other states at the public expense. What is the cattle owner doing in response to this paternal assistance to restrict tuberculosis. If we examine the annual reports of the Chief of the Cattle Bureau, Dr. Austin Peters, we find that in 1900 there were condemned, killed and paid for by the State 1423 head of manifestly tuberculous cattle; in 1901, 1341 head; in 1902, 1001 head; in 1903, 1172 head; in 1904, 1658 head; in 1905, 1625 head, and in 1906, 1202 head.

In other words, the state can buy as many clinically diseased cattle as it cares to appropriate money for. The cattle owner raises his tuberculous crop each year to be shouldered and paid for by public taxation. The only way to make any progress after these many years of patient compensation for diseased animals is for the state to take them without compensation, and for the public to patronize milk from tested herds. It has been amply shown that tuberculosis can be restricted if a little thought be given to the subject. Our national government distributes tuberculin and suitable directions for its administration, free of charge. Surely the means for individual initiative are not lacking, excepting one, and that is a little judiciously applied coercion. A law to remove the item of compensation after a definite number of years would probably bring about the desired reform.

There can be no doubt that the public is in a difficult situation with reference to the milk supply. The congested population of our large cities must draw its stock from a very large territory, and few can trace to the herd the milk that is daily delivered, owing to the multiplication of middle men necessarily engaged in its distribution and the distance from which it is brought. The adjustment of this difficulty is a delicate one and is an inter-state question. In the meantime, the helpless and distracted public may protect infants and young children from the occasional chances for infection by pasteurizing the milk. It should be stated in this connection that the lowest temperature which destroys tubercle bacilli is 140° F. and at this temperature the shortest allowable time is 20 minutes. The heating must be done in closed vessels to prevent the formation of a scum or film in which tubercle bacilli may survive the heat.

I wish finally to mention a possible source of infection to which no attention has been given. It is one which I mentioned as worthy of consideration in 1898. If the milk of tuberculous cows may infect children, may not the dust and dirt of cow stables affect adults working in them? I have already stated that only one not very clear case of bovine bacilli in sputum has been reported. It seems to me that the study of a series of cases of tuberculosis among those working in cow stables is desirable at the present time, and I am prepared to make such a study if the sputum is sent to me fairly fresh and a carefully prepared record of the history and present condition of the patient forwarded with it.

The conclusions which I think are warranted by the investigations upon human and bovine tuberculosis up to the present may be stated as follows:

1. The coughing consumptive is the chief source of infection.

2. The digestive tract is not the exclusive or even pre-

dominating portal of entry for pulmonary phthisis. It is highly probable that most cases are due to inhalation or aspiration.

3. In infants the bacilli probably gain entrance through all portals more easily than later in life, and the disease becomes more easily generalized. There are no rational grounds for believing that latency in infancy plays any appreciable rôle in the disease of later decades, but ingestion probably does play a much more important part in infantile tuberculosis, owing to habits and susceptibility of this period of life, than has been granted heretofore.

4. Bovine tubercle bacilli are found associated with a certain, at present not fully calculable, proportion of cases of abdominal and cervical lymph-node tuberculosis in infants and children.

5. There is at present no evidence that bovine bacilli may be transformed and assume the human type in the human body.

6. The discharge of tubercle bacilli into the milk of cows may take place abundantly in udder tuberculosis. In a small proportion of manifestly tuberculous cows without evidence of udder disease, it may take place at times in very small numbers.

7. A reasonable restriction of bovine tuberculosis below the danger limit to man is possible with the aid of tuberculin and segregation, and the removal of clinical cases and of udder disease. An eventually complete elimination can be brought about in this way in any herd, provided other domestic animals, such as pigs, cats and dogs are kept under control as possible reintroducers of the infection.

8. The complete elimination of bovine tuberculosis may be hoped for in the distant future. An active coöperation of individuals with municipal, state and national forces, by exerting a steady pressure and diffusing more knowledge among farmers as to the nature of tuberculosis and the use of tuberculin, may lead to its final disappearance.

9. The factor of bovine tuberculosis in the human malady is not of such importance that it should be permitted to imperil the proper execution of measures designed to relieve and cure the strictly human disease, or divert attention from it as the chief evil.

THE OUTLOOK CONCERNING A SPECIFIC THERAPY IN TUBERCULOSIS, AS VIEWED FROM THE EXPERIMENTAL AND COMPARATIVE STANDPOINT.

Having been asked to say a few words upon the present status and promise of serum therapy and vaccine in tuberculosis, I do so somewhat reluctantly and I place myself distinctly upon the experimental standpoint for two reasons. In the first place I have had no personal experience with the use of serums or vaccines as applied to cases of the disease. In the second place I believe that our fundamental data must come from experiments on animals.

Since the various kinds of treatment of tuberculosis may all be classed either as passive or active, I shall group my discussion under these heads. It is now pretty generally understood that serum therapy is a method of transmitting one or more anti-bodies, produced in some animal artificially, to the patient in order temporarily to fill a gap, to provide a substance which is wanting, but essential in the struggle against the disease. This want is filled by the serum until the body has sufficiently recovered to manufacture for itself the antibody. The use of serums is most successful in rapid toxic diseases in which there is only toxine to be disposed of, but no bacteria. When we come to the slower invasive diseases, like tuberculosis, the injection of a therapeutic serum would have to be made over long periods of time because its need would be continuous. But even then it is a question whether the serum would be of any permanent benefit because to throw off the disease the body must finally make its own anti-bodies. The serum is like a crutch

which must sooner or later be given up when the patient is strong enough to try walking alone.

Approaching the subject from the practical side we know that no serums have proved to be of any generally recognized value in the treatment of invasive diseases. The immunization of large animals is slow and difficult, and it can be carried only to a certain point. Such sera would in any event be very expensive and hardly within the reach of the average patient. Then again the repeated injection of foreign serum over a long period is not to be looked upon lightly.

From both the experimental and the practical standpoint the outlook for serum therapy is not encouraging, though I should not wish to say that it is hopeless. In some one form or another, unknown to us at present, there may be built up from the fragments of the present passive and active forms of treatment, a successful method accessible to all patients.

Owing to the difficulties both theoretical and practical surrounding the use of a curative serum, investigators have again turned to the method of inducing active immunity. This consists in stimulating and rousing the latent activities of the diseased body to produce its own anti-bodies. Begun with the introduction of the old tuberculin by Koch in 1891 it has had various fortunes, but it seems again fairly well established to-day. All the methods are based upon the introduction into the body of the products of tubercle bacilli. The fundamental underlying principle appears to be a reliance upon the diseased body itself with the understanding that it possesses sufficient forces to establish its own cure if these forces are properly husbanded and properly called forth. These forces are limited in amount at any given moment, and perhaps in time exhaustible. They should not be wasted because we know that the organism reacts with a very lavish production of anti-bodies. In the

treated horse for example, diphtheria antitoxin present in the blood can neutralize thousands of times the amount of toxin which is injected at any given time.

The presence of tuberculosis indicates that these anti-bodies are below normal, or have been so at some time in the past. These considerations suggest the use of very small doses of the stimuli and only at the proper time. Sir A. E. Wright maintains that in the anti-bodies known as the opsonins we have such a guide, and the index of this guide is phagocytosis. Some are inclined to neglect this guide, and utilize clinical symptoms in the treatment.

It follows from the theory of action of these stimulating substances that they are not called for in active, febrile stages, when the body is apparently fully roused and needs no stimulus from without. Under such conditions an anti-toxic serum might be helpful to neutralize the over abundance of toxins until the system had recovered itself.

It is furthermore evident that the application of these substances may bring into action various tissues of the body besides the immediate focus of disease. The whole body may thus be laid under contribution artificially to relieve a limited area, which is unable to protect itself.

In the practical development of this mode of treatment by gradual cautious stimulation, many substances have been used, all of them derived from the tubercle bacillus. The old tuberclin comes first in the order of time, next the new tuberculin, representing ground-up bacilli. Then there are the watery extracts of tubercle bacilli used by Maragliano, the filtrates of the culture fluids as used by Denys, and lastly the mystical tulase of Behring. Whatever may be the perfection to which these methods of active immunisation may be carried in the future, there still remains the unknown quantity of the capacity of the individual patient to produce the necessary anti-bodies and bactericidal forces. If these are lacking therapy is powerless to check the disease.

This discussion, brief though it be, has led us up to three propositions:

1. Serum therapy is at best a temporary palliative method in infectious diseases which are long drawn-out, and which must finally be met by the specific resources of the body itself.

2. The need of discriminating power on the part of the practising physician between individual and individual in the application of remedies which stimulate the production of anti-bodies. He is evidently not going to be eliminated by an advance in medical science. In fact he must be better trained than ever, especially in the fundamental principles of immunity, — a subject quite new but already rich in acquisitions, which may be called the physiology of the infectious diseases.

3. To each human being are given certain talents to husband and develop and not to squander, for therapy cannot add to them. It falls to the lot of the physician to discover during disease the size of those hidden talents if he can, and to utilize and manipulate them in the best interests of his patient.

ARTICLE XXII.

THE ORGANIZATION OF TUBERCULOSIS CLASSES.

By JOSEPH H. PRATT, M.D.
OF BOSTON.

READ JUNE 11, 1907.

ON THE ORGANIZATION OF TUBERCULOSIS CLASSES.

THE Emmanuel Church Tuberculosis Class was organized July 1, 1905. Since that time fifty-four consumptives have been members of the class. Seventeen have already recovered and returned to work. The disease has been arrested in seventy-five per cent. of the incipient and moderately advanced cases. The first three members who joined the class within a few weeks of its formation have been at work over a year. Although they had advanced tuberculosis the disease became entirely arrested, and they were restored to their full working capacity.

A description of the class method of treating pulmonary tuberculosis will appear in the next volume of the Transactions of the National Association for the Study and Prevention of Tuberculosis.

Three additional tuberculosis classes have been formed in Boston. The Suburban Tuberculosis Class was started by Dr. John B. Hawes in January 1906. It was designed for patients who lived too far from the city to be visited by the nurse of the Emmanuel Church Class. The Arlington Street Church Tuberculosis Class is under the charge of Dr. N. K. Wood. It has been in existence over a year. Recently a class intended principally for Jewish patients was started by the Mt. Sinai Dispensary. A friendly visitor was selected who could speak Yiddish. The Cambridge Tuberculosis Class was organized by the Cambridge Anti-

Tuberculosis Society in 1906. About six months ago classes were started in New York, Philadelphia, and Baltimore, and more recently in Lynn and Brockton. Steps have been taken to form classes in Washington, Pittsburg, Troy, and Los Angeles.

There are several advantages of the class method of home treatment over sanatorium treatment. It is more economical. There is no expensive plant to be erected and maintained. The money appropriated can be at once utilized for the care of consumptives. If the patient regains his health in his own climate and in his own home it is easier for him to keep up the hygienic life after he has returned to work. With a class of small size the physician and nurse can give more attention to the individual patients than is possible in most sanatoria. Early cases in other members of the family are more readily detected. The educational effect on the patient's friends and neighbors is greater.

The disadvantages are equally apparent. It is easier to secure the necessary control so essential to successful treatment in a sanatorium where the patients are always under the eye of the directing physician and the nurse. Better food and better care can usually be given a poor patient in a well managed sanatorium than in his own home.

In organizing a class the first task is to secure its adequate financial support. The Rev. Dr. Worcester of Emmanuel Church has raised about $1000.00 a year for our class. This has been obtained by Sunday collections and by individual gifts. The first grant of $500.00 was obtained from a fund known as the Parish purse which is placed at the disposal of the rector. Facilities for leading the out-of-door life such as tents, reclining chairs and blankets have been provided and medical care and the services of a paid nurse furnished.

We have not attempted to treat homeless and destitute consumptives. Some of our members have been aided by

small allowances, and in a few instances board and room rent have been paid. It has been a constant aim to place the responsibility of the support of our members on their own families. Our work could have been made more efficient not so much by increasing our membership as by increasing the financial aid to some of our poorer members during treatment and during the period after recovery while suitable work was being sought. One of our class to whom we made an allowance of $1.00 a week and who also received $2.00 weekly for a short time from a Jewish charity was so pressed for money that he was forced to pawn his watch and his wife's wedding ring. Knowing the small amount of money in our treasury he took this step rather than apply to us for more aid. A graduate with no resources and a child dependent upon her was forced to accept a situation as waitress in a restaurant where the hours of work were from five in the morning until ten in the evening.

Dr. F. T. Fulton secured the necessary funds for starting the Providence Class from the Social Service Club of the Central Congregational Church of that city. He arranged for a representative of the Emmanuel Church Tuberculosis Class to give an account of the class method and the results obtained at the church. A series of lantern slides were shown and charts and records kept by the members of the class were exhibited. After this demonstration the Club voted to appropiate $500.00 for the work.

A Boston woman, not connected with Emmanuel Church, became interested in the movement and arranged a small home meeting to which four of her friends were invited. After the method of work had been explained these five ladies subscribed $500.00 for the formation of a new class; the success of which in the hands of Dr. N. K. Wood was such that at the end of six months the Arlington Street Church was willing to assume the maintenance of the class. Dr. J. B. Hawes secured both money and volunteer visitors

for the Suburban Tuberculosis Class by publishing an appeal in the Boston Transcript. Ex-Governor Douglas announced the day after the formation of the Brockton Tuberculosis Class that he would finance the enterprise and gave at the same time $1000.00.

A young woman in New York raised among her friends sufficient money to start the first tuberculosis class in that city. It has already done a remarkable work. The director, Dr. W. L. Niles, has demonstrated that tuberculosis can be successfully treated by the class method under unfavorable conditions in the crowded sections of New York City. Dr. David Riesman's class in Philadelphia has been supported by one gentleman whom he interested in the movement. The expenses of the Baltimore class have been largely borne by Dr. Dencker, who in addition has given his entire time to the work. In Lynn circulars calling attention to the class method were sent out and by this means sufficient money was obtained to start a class. In Troy a society for the home treatment of tuberculosis was established under the patronage of influential men. Many people became interested, so that $1200 has already been secured by five and ten dollar subsciptions. A copy of the circular issued by this organization will be found at the end of this paper.

These facts are presented to point out the various ways that classes may be supported.

The co-operation of all the local charitable, religious, and fraternal organizations should be secured. Although in a large city such as Boston a class may be maintained by a single church it will be the better plan in most instances to have the work started under the auspices of some organization that can look for support to the entire community.

Failure will certainly result unless the services of a physician and friendly visitor are secured who are familiar with every detail of the modern treatment of consumption. Our

work would have been impossible if we had not had from the beginning the services of a paid friendly visitor. She was expected to devote half of each day to the work. As a matter of fact our friendly visitor, Mrs. Oreon, has frequently insisted on working many additional hours. Our success has been chiefly due to her tireless devotion. I recently learned that she called daily for two weeks on one patient before she could induce him to sleep out of doors. He was an ignorant man and his friends and relatives warned him that death would surely follow if he made the attempt, as it was mid-winter. He has been sleeping in the fresh air for over a year and has not only regained the sixty pounds he had lost but the disease has been arrested and he has returned to work.

It is not necessary that the friendly visitor should be a trained nurse. Constitutional energy, tact, and sympathy are the essentials. There have been several notable instances in which the work of friendly visitor has been most successfully carried out by young women who have had no hospital training. Volunteer visitors can be of great assistance. We have had two volunteers who, working with the friendly visitor, have rendered efficient service in the solution of the social and financial problems. The social aspects need to be developed. They are quite distinct from the medical side. Students of sociology should join heartily with medical men in this work. The medical direction should be in the hands of one physician. He should be paid a salary sufficient to enable him to devote a fair portion of his time to the class and make a special study of the disease he is called upon to treat. A few weeks spent at Saranac or at the Phipps Institute in Philadelphia would be invaluable. Those in charge of the Boston classes will be glad to give instruction in the class method. It has been found that the tuberculosis classes have furnished a good opportunity for the physicians connected with them to ac-

quire a first class clinical knowledge of pulmonary tuberculosis.

Much depends on the selection of cases. The fact that all of our nine incipient cases have recovered while seven of the ten far advanced cases have died indicates the value of early diagnosis and prompt treatment. Treatment is never begun too early, it is often begun too late. For the reason that the disease may be so insidious in its development and because recovery is not only more sure but more rapid when the patient is at rest, we have insisted that all of our patients should cease their work and devote themselves entirely to the task of getting well.

The present wave of optimism in regard to the treatment of consumption has its dangers. Many physicians make the fatal mistake of minimizing the seriousness of the disease in its early stages both to themselves and their patients. I am often asked by physicians to treat in our tuberculosis class consumptives "who are not sick enough to give up their work" or "who cannot afford to stop working." I am glad of this opportunity of pointing out to the members of The Massachusetts Medical Society their great responsibility in this matter. To let a man continue at work after the diagnosis of active pulmonary tuberculosis has been made is as foolish as it would be to let a fire burn unchecked until it became serious before calling the fire department. Doctors and social workers frequently speak of their inability to persuade consumptives to leave their work and take the rest treatment. This failure is usually due in the first place to the consumptive's ignorance of his true condition and secondly to his lack of financial resources. We have overcome these obstacles by telling him the truth, and by promising that if he will join the class, keep absolutely quiet, sleep out of doors and follow our guidance in all things we will see that the ways and means are furnished.

When a suspected case is submitted to us a definite diag-

nosis is always made before treatment is instituted. If tubercle bacilli cannot be demonstrated in the sputum, the tuberculin test is tried. I have seen no harm result from tuberculin in the hundred or more cases in which I have used it. The initial dose I employed is $\frac{1}{10}$ of a milligram. If there is no reaction a second injection of 1 milligram is given, and if necessary a third test with 10 milligrams is made. The details of preparing the dilutions will be found in the recent paper of my associates, Dr. Hawes and Dr. Floyd.* After the diagnosis has been made the patient is told that he has consumption and if unwilling to heed our advice the seriousness of the disease is impressed upon him. In the old days I have frequently overheard a conversation somewhat as follows between the hospital externe and a patient in whose sputum tubercle bacilli had just been discovered. "We find a little trouble in your lungs," began the doctor speaking rather hesitatingly, "I don't suppose you could give up your work for a few weeks and go to the Rutland Sanatorium, could you?" "No," said the patient, "I have a family to support and no money." "That is too bad," said the doctor, "it would be better if you could stop work and go to Rutland, but as you cannot do that be sure and stay out of doors all you can, eat plenty of food, take this medicine for your cough and come back and see me in two weeks."

I never saw cures produced by this method, and my experience accords with that of my colleagues.

Since the tuberculosis class was established I cannot recall a single instance in which we have failed to induce a man of fair intelligence and good habits to stop work and to take the treatment. If a man tells me he has a wife and children dependent upon his weekly earnings and no money on hand I advise him to explain the situation to his relatives. Often there is a brother or a brother-in-law who is

* Boston Medical and Surgical Journal, 1907, CLVI, p. 694.

earning fair wages. I try to impress the following facts upon the sick man. The disease has its clutches upon him. Soon his strength will begin to fail and within a few months in all probability he will be too ill and weak to work. Then he will tax the resources of his family to the utmost. Often some one who might be earning money must remain at home to care for him. In addition to the expenses of his illness there is the increased financial burden attending death and burial. After the breadwinner is dead, provision must be made for the wife and children, not for months, but for years. It is cheaper for the consumptive to get well than to die. This plain speaking may seem brutal, but it is often the means of saving life. It is a line of argument that rarely fails with the relatives. If they refuse to loan money, we have been able to secure from other sources the necessary funds to enable the patient to take the treatment.

In Oxford, Providence, and Brockton several large manufacturers have provided free treatment in sanatoria and at home for consumptives in their employ. Dr. F. T. Fulton of Providence has found that those men who began treatment before constitutional symptoms had developed were able to return to work within a few months. Among these there has been thus far no recurrence of the disease.

Our greatest need in the Emmanuel Church Tuberculosis Class is provision for the exceptional cases who need prolonged financial aid, and for the after-care of our graduates. Many difficulties financial and sociological are presenting themselves. The medical problems have been largely solved. Much could be accomplished by giving a small allowance to the individuals in whom the disease has been arrested while they are seeking suitable employment. In Bardswell's valuable monograph on the Consumptive Working Man will be found the report of an after-care committee as it is called which was organized by the Kelling Sanatorium at Holt, Norfolk. This committee sent a printed

leaflet to employers of labor, a copy of which will be found appended to this paper. There is urgent need that similar committees be formed in this country. In England employment was furnished chiefly by people with gardens and by farmers. Light work in-doors is better for a consumptive with arrested disease than heavy work out of doors. If obliged to spend the day in-doors the individual with arrested disease should not fail to sleep in the open air.

The establishment of a loan-fund for the benefit of individuals of good character in the curable stage of the disease would be of great economic and philanthropic value. The administration of such a fund might be quite independent from that of the tuberculosis class. In some cases the issue between life and death depends upon whether or not there is an available fund of a few dollars a week. Our patients are of entirely different type from those familiar to charity workers. In the majority of cases the disease had been the indirect result of over-work. They are thrifty and are able to live comfortably on little money. The fact that they carry out the class treatment faithfully for weeks and months indicates that they have perseverance. If such a fund is established loans should only be made after the needs of a case have been most carefully investigated. As only a limited number of cases can be helped at first preference should be given to those who can be most quickly and surely returned to the ranks of bread winners. Alcoholics should be viewed with suspicion. An intemperate man under our care who made speedy and marked improvement went on a debauch with the first money he earned after returning to work. Experience with young married women is also disappointing, for the reason that pregnancy and child-birth are so apt to be associated with a lighting-up of the disease.

If there are children in the family and the resources are scanty it is well to find temporary homes for them in the

country, especially if the mother is tuberculous. In this work we have been greatly aided by the Children's Mission. Not only does removal of the children lessen the difficulties of caring for the family, but it often saves the children from becoming victims of the disease. We found unsuspected cases of tuberculosis among the children in no less than twenty per cent. of the families represented in the class at the present time.

Attention should be called to the establishment in New York of a special employment bureau for the handicapped.*

If an after-care committee be formed in connection with our tuberculosis classes it is probable they would proceed on somewhat similar lines. An effort should be made to interest the employers of labor in this work. The positions of watchmen or care-takers should be specially sought for our graduates. Honest, sober, and reliable men could be furnished.

Most of our graduates have been able to return to their former employment, and yet maintain their health. If arrest is secured in advanced cases only light work under favorable conditions should be attempted. It is even more difficult to secure suitable work for women than for men.

A specific instance of the difficulties that confront us may be cited. In November, 1906, a type-setter entered the class with incipient phthisis. He owned a little property in England, and although he earned fair wages had saved no money. His wife was a nervous invalid unable to take boarders, and there were two children to be cared for. We turned to Mr. A. M. Wilson, the efficient secretary of the Boston Society for the Relief and Control of Tuberculosis, and he was able to obtain a weekly loan of $10.00 for a period of about two months. The property in England was not regarded as good security, and he was unable to

* See the interesting paper by T. C. Janeway and C. C. Carstene in *Charities*, February 3, 1906.

dispose of it. After this relief failed a lady in the Emmanuel Parish loaned the same amount weekly for four months longer. The disease in the lungs became completely arrested. About six weeks ago, just as he was on the point of returning to work, symptoms of tuberculosis of the spine developed. For nearly a month he received free treatment in the wards and convalescent home of the Massachusetts General Hospital. Last week we were told by the orthopedic surgeon to whom we referred the case that an apparatus to hold the spine immobile must be worn for three months more. The outlook for complete recovery is good. He feels well and strong, but so long as he is obliged to wear the plaster cast he will be unable to do his work as a "job compositor." The printing firm for whom he worked have held a place for him. What shall be done in this case? There is the rent to be paid and the family to be fed. At the last meeting of the class the friendly visitor reported that there was no food in the house and he had to borrow a nickel for his car fare from a neighbor. I am glad to report that he received this week $10.00 from the class fund. Only temporary aid can be furnished from this source. Inasmuch as the total amount of money at our disposal is not over $20.00 a week it can readily be seen that we cannot pay out $10.00 weekly to one individual for any length of time. What better memorial for a relative or friend could there be than the establishment of a loan fund for cases such as this.

To show what can be done with a small amount of money the case of a young seamstress may be cited. She consulted a physician in the spring of 1906 on account of a cough. He found tubercle bacilli in her sputum and referred her to our class. She had only $50.00, and her parents who lived in Nova Scotia were too poor to help her. She was boarding with a friend who charged her only $2.50 a week. We gave her this amount of money weekly for

four months and loaned her a tent and reclining chair. She enjoyed the privileges of the Parker Hill Day Sanatorium where lunches and a good dinner were furnished free. She entirely recovered and returned to her home in Nova Scotia for the winter. She writes that she has been perfectly well since graduating from the class and expects to return to work soon. Who can doubt that the loan of $40.00 saved her life ?

The social and economic aspects of this movement are assuming increasing importance. Is it not possible that in the practical work of the tuberculosis crusade the sociological and medical departments of our universities may combine ? The churches with their organized forces might furnish great aid if they were awake to their opportunity.

Although it has been demonstrated that the class method furnishes an economical and efficient means of treating tuberculosis in the homes of the poor, three important points should be borne in mind if the tuberculosis classes are to be successful. First: they should be directed by physicians familiar with the details of the modern treatment, and the right women should be selected for the position of friendly visitor. Second: there should be adequate funds for the support of the class. Third: every physician in the community should learn to recognize the disease in its early stages and to refer the cases for treatment without delay.

APPENDIX.

Copy of the Circular issued by the After-Care Committee of the Kelling Sanatorium.

To Employers of Labor and Others Interested in the Working-Class Consumptive.

It is felt that it should be more generally recognized that, in dealing with consumption, sanatorium treatment is only the first step.

Of suitable cases, treated at a sufficiently early stage, a large proportion are discharged fit for work, but if the good gained at the sanatorium is to be maintained, it is essential that the patient should, on leaving, be able to go to suitable employment in suitable surroundings.

The Committee of the Kelling Open-Air Sanatorium, feeling the vital importance of this side of the work, have appointed a special sub-committee called the After-Care Committee, who consider the future of each of the sanatorium patients, and, where necessary, endeavour to obtain suitable work for him when he leaves such employment and surroundings.

Work is provided for a certain number at the sanatorium itself, but as it is impossible to provide in this way for more than a very small fraction of those discharged, the committee now venture to appeal to employers of labour to assist them.

They can help, it is suggested, in two ways: —

1. By being willing now and then to find temporary light work for a man.
2. By occasionally giving one of our men the chance of a suitable permanent position.

In the first case the idea would be to enable the man for a short time to earn a bare living wage in suitable surroundings while he looked about for permanent work, and, in the second, to provide him, at no doubt a moderate wage, with suitable permanent employment.

Our men are almost all skilled in some trade, and we would obtain in every case a character from the previous employer.

It would be clearly understood that no responsibility whatever as to the man's health should be undertaken by the new employer; if a man so employed should break down, the committee would like to be informed, and would do all they could to secure him further treatment.

The employment may be either out of doors or in pure air indoors. It should not involve any great physical effort, nor should it require to be done against time or at high pressure.

The following list contains a few of the employments suggested: —

Light work about a farm or garden.
Driving.
Caretaking.
Agency work, as for instance, insurance agency, rent collecting, or some form of travelling, etc.
Clerical work if in good conditions.
Estate work, such as light carpentering, looking after an engine, etc.
Motor car driving.
Check taking.
Door keeping, and many others.

The risk of infection has been mentioned as an objection, but there is no doubt that it has been greatly exaggerated, and, in any case, it is certain tha a man who has been treated at a sanatorium, whose symptoms have disappeared, and who has been taught the precautions, is not only unlikely to be a source of infection, but is actually a missionary of health to all with whom he comes in contact.

The preferential consideration given to subscribers with respect to the admission of patients into the sanatorium will be given to any employers who will consent to give, instead of a subscription in money, this invaluable practical aid.

May we write to you when we want employment for a man? Please reply to — the Hon. Sec., Kelling Open-Air Sanatorium, Holt, Norfolk.

COPY OF THE APPEAL FOR THE SOCIETY FOR THE HOME TREATMENT OF CONSUMPTION IN TROY.

> "We must care for the consumptive in the right place, in the right way, and at the right time until he is cured; instead of, as now, in the wrong place, in the wrong way, and at the wrong time until he is dead." — PRYOR.

There has been, during the last few years, a great revival of interest both among physicians and business men in the question of consumption. Everyone is coming to realize that this plague is the most dangerous of any that we have to contend with.

It kills, every year, more people than any other disease. Last year there were 240 deaths from consumption in Troy, an average of 20 each month. The great majority of the deaths were among the poor between the ages of 20 and 40 years, the time of life when men and women are most essential to the support of their families and of their children. They cannot go away or receive the benefits of treatment in a sanatorium, therefore they must be cared for at home.

In many cities of the United States measures have been taken to fight this disease and the results have been remarkable. We are going to fight consumption in Troy and we ask for your interest and assistance.

A class is to be formed of consumptives who cannot afford to leave home to go to a sanatorium or to pay for continuous medical care. The Society has secured the voluntary services of Dr. H. W. Carey as physician-in-charge, and Dr. H. C. Gordinier and Dr. J. B. Harvie as consultants.

The class will be under the care of the physician and a nurse who will instruct the patients in the three essentials for recovery — *Rest, Living Out of Doors, and Proper Food.*

The class meets once each week for examination by the physician, and each member is visited at the home by the nurse, who will see that the instructions are properly carried out. This is the method used so successfully in Boston, Providence and New York, where, in most instances, the progress of the disease has been arrested, in many instances cured.

The cost of maintaining the Class is $50 a patient a year, the expenditure being limited strictly to the purchase of tents, cot-beds and reclining chairs to enable the patient to live comfortably out of doors and to pay for the services of the nurse. In order to meet the expenses of carrying on the work for a year we must have $1,000 in hand and we ask your aid by a contribution of $10.

(Signed) PAUL COOK,
FRANK E. HOWE,
EDWARD MURPHY, 2d,
GEORGE ALFRED CLUETT,
LELAND THOMPSON,
EDGAR H. BETTS.
Committee.

DISCUSSION.

Dr. JOHN B. HAWES, of Boston: I have been associated with Dr. Pratt right from the start, and from the very beginning I was tremendously impressed with what this class method can accomplish. There is one thing which he has not mentioned, and which I think is one of the principal factors in successfully handling these cases, and that is, the personal element. Every one of these patients who have come here today is a personal friend of mine. They are being taught how to help themselves. As Dr. Pratt's class increased the need of another class was apparent, and there was another started at the Massachusetts General Hospital under my charge, and this was called "the suburban class," of which there are now three. The patients sleep out of doors wherever possible, and come to the hospital for the class work once a week when they are able, where we talk over the details of the past week. The first patient shown here by Dr. Pratt had been under my care at the hospital for six weeks, and nothing seemed to be accomplished under the old out-patient system until Dr. Pratt commenced this system of personal supervision

of the patients in their homes, and to the hard, individual personal work which he has put into it, I think the credit is largely due.

Dr. I. J. Clark, of Haverhill: A year ago there was a committee appointed in Haverhill to look into this matter, and to further the treatment as much as possible. This committee included members from Lawrence, Newburyport and our own town. I am very glad to report that we have organized a society in Haverhill, and solicited funds from our people. We have officered it with about all the doctors and clergymen, and I have patterned as far as possible after Dr. Pratt's method. It does not seem practicable to have a class at present, but I have suggested it to the supervisor. I intend to work with him in carrying out the details. We are also to have the State exhibit. It seems to me a movement which every town of any size should take up. It is surprising to see how much interest in this is shown by the public. I do not think the matter will lack for means. I am very much interested in this matter, and will say that we find it very difficult to carry out the rest treatment. I had a patient recently who by careful supervision and treatment had become much better with no fever, but he insisted on going, camping out and fishing trips, and he had a relapse. He did very well, however, under the treatment, while he remained quiet.

Dr. David Townsend, of Brookline: There is one phase of the work which has not been mentioned, the "Day Camp." The Boston Association for the Relief and Control of Tuberculosis has operated such a camp the past two summers on Parker Hill in Roxbury through the courtesy of the Robert Brigham trustees. This summer the Camp has been established on the estate bought for the proposed Municipal Consumptive Hospital, through the courtesy of the municipal hospital trustees. The camp is modeled after those in Germany, particularly those in Berlin. The patients come for the day only, they arrive early in the morning and go home late in the afternoon. They are given two lunches, one at ten, the other at four o'clock, and a dinner in the middle of the day. Each patient is examined once a month, and weighed once a week; his pulse and temperature taken twice a day. The aim of the officials of the

Camp is to know each patient intimately, and to encourage him to confide in them. His day at the camp is regulated and every thing done to remove all worry and anxiety. He is kept under careful supervision. Such a camp may be readily attached to a Hospital, and can be run at a moderate expense as has been proved by the Good Samaritan Hospital, in Boston, the past year. A Day Camp is of value as it removes for a time the source of infection from the community and the home; it enables some cases to obtain admission to a sanatorium which could not otherwise go; it enables cases to obtain proper care which, although suitable, for some reason cannot go to a sanatorium; it obviates in some cases the necessity; it helps to complete the cure in some cases which have been discharged from a sanatorium. It is also a value on educational lines. As to the permanent good which results I can only say that of the cases present at the Camp in the season of 1905, one-third are able to work all or a part of the day. One point which I would like to emphasize is the importance of a complete examination of all the members of a family in which there is a tuberculous patient. In this way we are enabled to detect the disease in the earliest stages, and to take proper steps to prevent its spread.

Dr. A. C. Getchell, of Worcester: One other point should be touched upon at this time it seems to me. It is the matter of ways and means. Dr. Pratt's results are very encouraging and his work is of great value. Can others in other places do as good work? He has the material resources of a large church and a well supported tuberculosis organization at his disposal. Looking to our work in Worcester we have neither; nor, so far as I can see, are we likely to have. On the other hand it is quite within the range of probability that one of our large hospitals may soon make proper provisions for poor consumptives. As a practical measure I think we will sooner provide suitable care for these unfortunate persons by putting our main energies upon utilizing the equipment of existing institutions with such slight additions as may be necessary. As has been said, Dr. Pratt's personality is an important element of his success. While this has contributed so largely to his work it cannot be counted on with definiteness in

other places. To my mind the great value of his work, and I sincerely hope he will keep on with it, is a demonstration of what can be done at home with the moderately poor consumptives. But I feel that we shall make no material progress in caring for the very poor, the most dangerous class of all, until we provide adequate institutional care for them.

DR. C. S. MILLET, of Brockton: One word about the personality of Dr. Pratt, about which we have heard so much this afternoon. If there is anything in taking a real heart-felt interest in your patient, in paying attention to detail and insisting upon entire obedience on the part of the one most interested, which the rest of us can't imitate, then, of course, Dr. Pratt's personality is certainly the most important factor in the kind of work which he has originated.

Otherwise there is nothing about this work that any man here can't do.

ARTICLE XXIII.

MEDICAL INSPECTORS: THEIR FUNCTION.

By CHARLES HARRINGTON, M.D.
OF BOSTON.

READ JUNE 11, 1907.

MEDICAL INSPECTORS: THEIR FUNCTION.

YESTERDAY there was passed by the Senate to a third reading, a bill, which already has passed the House of Representatives, entitled "An Act to Provide for the Establishment of Health Districts and the Appointment of Inspectors of Health."* The bill was first reported in the House on May 1, by the Committee on Public Health, after full consideration of so much of the annual report of the Chief of the Massachusetts District Police as relates to the manufacture of clothing in tenement houses; so much of the Governor's address as relates to medical examination of minors in factories; a petition for legislation relative to the spread of tuberculosis; a petition for legistation to provide for the transfer of certain powers and duties of the inspection department of the District Police to the State Board of Health; a petition for legislation to provide for proper sanitation, ventilation and light in factories and shops and for the appointment of medical inspectors; a petition for legislation to prohibit overcrowding and bad air in factories and workshops; and a petition that medical inspectors be appointed throughout the Commonwealth.

Should the bill become law, it will be the duty of the State Board of Health to "divide the Commonwealth into not more than fifteen districts, to be known as health districts, in such a manner as it may deem proper and necessary

* The bill was enacted in both houses, and was signed the Governor on June 19.

for the carrying out of the purposes of the (this) act"; and, this having been done, it will devolve upon the Governor to appoint in each such district "one practical and discreet person, learned in the science of medicine and hygiene, to be state inspector of health in such district." The appointees will hold office for five years, but may be removed by the Governor and Council at any time.

The duties of the inspectors will be so varied and extensive as to make the positions anything but sinecures, and in most of the districts their proper performance will demand of the incumbents practically their entire time and will involve constant travel from place to place. The bill provides in the first place that each inspector "shall inform himself respecting the sanitary condition of his district and concerning all influences dangerous to the public health or threatening to affect the same." This alone will be no small task, for although the area of the Commonwealth, in comparision with that of almost all other States of the Union, is small, the fifteen districts together will include 33 incorporated cities and 321 towns, and each district, therefore, must contain a considerable population, divided among not a few distinct communities, large and small, urban and rural, manufacturing, agricultural, residential, and mixed. In order, therefore, to inform himself and keep informed as to the sanitary condition of his district, and to carry out the duty with which he is specifically charged in the next clause, to "gather all information possible concerning the prevalence of tuberculosis and other diseases dangerous to the public health within his district," he must keep in touch with all of the local health authorities thereof, watch the incidence of communicable diseases in each city and town, inquire into their causes, be prepared to suggest preventive measures, and to "take such steps as, after consultation with the State Board of Health and the local health authorities, shall be deemed advisable for their eradication."

Impressed with the value of the medical inspection of school children, the attention of His Excellency the Governor was drawn to the advisability of extending the same protection to minors employed in factories, under conditons, which, in very many cases, are far more inimical to health than the conditions to which those confined for shorter daily periods in schoolrooms are subjected; and the matter was made the subject of an earnest recommendation in his inaugural address. If the bill becomes law, it will be the duty of each inspector to have an eye on all employed minors, to examine them as to their health, and, whenever advisable or necessary, to call their ill-health or physical unfitness to the attention of their parents or employers and of the State Board of Health. In some districts this duty will make large demands upon the inspectors' time, for in many manufactures large numbers of minors find employment in rooms where they are exposed to influences which tend to break down their resistance to disease, and especially to tuberculosis.

A third duty imposed upon the inspectors is that of enforcing, under the direction of the State Board of Health and in place of the inspection department of the District Police, the provisions of existing laws relative to ventilation, light and cleanliness of factories; to providing suitable seats for women employed in manufacturing, mechanical, or mercantile establishments; to the employment of minors in the manufacture of acids; to proper water closets for factories and workshops; to defective sanitary arrangements, drains and water supplies; to nuisances; to cleanliness, ventilation, and provision for water closets in public buildings and schoolhouses; to manufacture of clothing in dwelling houses and tenements; to providing pure water for employees; to installing hoods, suction pipes and fans or blowers where dry grinding on emery wheels is carried on; and to the maintenance of suitable dressing rooms and

wash rooms in foundries. In other words, every manufacturing establishment, every public building and every schoolhouse will be under the sanitary supervision of the health inspector of the district in which it lies, and it will be the duty of that officer to visit each such place with reasonable frequency, to report upon its condition and to enforce the law relating to any defect or omission.

In addition to the duties specifically mentioned in the bill, the inspectors will be required to perform such other duties as the State Board of Health — under whose general supervision they will be — may from time to time determine. They will be required to keep records of their proceedings and observations, to submit annual reports to the Board, and from time to time to furnish the Board with information concerning conditions affecting the public health in their respective districts; and whenever they send written suggestions to local authorities they shall send copies thereof to the Board.

From this brief resumé, one may well understand that for the proper and adequate performance of the various duties imposed upon them the incumbents of the positions must necessarily be men of good medical training and experience, of good physique, for the work will be arduous, of good judgment in sanitary matters, and endowed with that most necessary qualification, tact.

DISCUSSION.

Dr. H. L. CHASE, of Brookline: I only rise to express a hope that members of the profession will make suggestions to the Governor as to the appointment of suitable men for these very important offices of inspector.

Dr. W. J. SLEEPER, of Westford: I endorse what Dr. Harrington has said, and it seems to me that this matter of factory and health inspection should very properly be

under the supervision of the Board of Health, and not under the District Police as it has been so many years. It is not the function of the police, and it has not always been carried out in the wise way it should be. While their intentions were good they lacked the necessary qualifications that scientific knowledge would give.

In answer to Dr. HENRY JACKSON'S question as to whether much of the work could not be done by non-medical men, Dr. HARRINGTON answered: An effort was made to induce the Committee to strike out the word "medicine" and insert in place thereof the word "sanitation," but inasmuch as the work of the proposed inspectors is very largely medical and requires medical training, the Committee declined to make the change and decided that the word "medicine" should be retained.

It is true that some of the work which the inspectors will have to perform can be done by others than graduates in medicine, especially that having to do with inspection of factories. As a matter of fact, the graduate of an engineering school is likely to be a better judge of ventilating apparatus than the average medical man. The medical man knows when ventilation is defective, but may not know how to direct changes to bring about improvement. Again, it does not require a man skilled in medicine to point out that a given atmosphere is overcharged with dust and fumes. On the other hand, one of the most important duties of the proposed inspectors is to examine into the health of all minors employed in factories. Such an examination is beyond the power of the most gifted engineer, unless he has had a medical training.

It must be understood that this bill was based upon a large number of petitions endorsed by a number of different interests. In the first place, there were those who sought to take the factory inspection out of the hands of the District Police, and prominent among these were leaders of the labor unions, who asserted before the Committee that until the inspection of factories was taken out of the hands of the District Police and put under men with scientific training they could not hope for sanitary improvement of factories. Other bodies interested were those working for the suppression of tuberculosis, and it will be noted that this disease is specifically mentioned in the bill, it being one of the

duties of the inspectors to inform themselves concerning the prevalence of tuberculosis and other diseases dangerous to the public health, and to suggest methods of combating the same. There were others who for many years have wished to have the State divided into health districts, with medical officers of health such as are employed in a number of states, and in Great Britain and other foreign countries. Attempts so to divide the State have hitherto met with no success, and it would probably have been absolutely impossible to secure legislation appointing medical officers of health without a combination of interests such as came into being this year.

With reference to the point that it does not require a medical man to do some of the work of the proposed inspectors, let me call attention to the fact that there is in the bill a clause which empowers the State Board of Health to employ such sanitary experts as may be needed: this has reference to ventilation and other matters involving a knowledge of engineering.

Dr. H. L. DEARING, of Braintree: In connection with what Dr. Harrington has said, I wish to say that in Braintree the inspection of the schools and factories has been carried on by three public spirited physicians, and from our experience it seems very plain to us that the medical man is the only man who is fitted to make these inspections properly. In our investigations we have found that the floors of the factories are in very bad, in fact often filthy condition. Before inspection the floors were washed up twice a year, now they are washed up twice a week. The danger of drinking out of the same cup is well illustrated by this example which I will cite. One of the large employers of the town came to me with a sore on the lip, which he and I also felt sure came from using the same cup as the employees. He told me that after that he would see that there was proper medical inspection of his factory.

The Chairman, Dr. A. T. CABOT, of Boston: It seems to me that it is not only the duty of the members of the Associated Committees to recommend to the Governor capable medical men to serve as inspectors, but that it is also their duty to ally themselves with the inspectors when appointed, and to assist them in every possible way.

ARTICLE XXIV.

FACTORY INSPECTION.

By FRANK G. WHEATLEY, M.D.
OF NORTH ABINGTON.

READ JUNE 11, 1907.

FACTORY INSPECTION.

LEGISLATION to protect the public health has for many years been the settled policy of this Commonwealth. Every year the legislature enacts new laws or amends old ones. While in many cases changes in law have not notably improved conditions still the general trend of legislation has been helpful. Perhaps the last two or three years have been more than usually fruitful in protective legislation. The legislation leading to the recent investigations of health conditions in industrial life by the State Board of Health has been of especial value, largely because of the intelligent methods of investigation adopted by the very able and efficient secretary of the Board. Competent men were selected to do this work and the commercial side of the problem, that is the employers' interests, were duly considered. As a result of these investigations we have a certain amount of valuable information that may be made a basis for a more intelligent appreciation of the problem. The conditions discovered were, in many cases, fairly good, but in a discouragingly large number distinctly bad, and on the whole the theory that a large number of municipalities have local boards of health, either incompetent or unwilling to do their duty, seems to be established. A necessary inference from this condition is that it is incumbent upon the Commonwealth to in some way come to the rescue, and it is hoped that pending legislation, of which it may be premature as yet to speak, may accomplish a great deal along this line. Factory inspection as a phase of the general health question is every year becoming more important.

This because not only is the total number of factory employees increasing, but the small workshop employing comparatively few people, is giving place to the mammoth plant where thousands work under the same roof.

I propose to consider briefly the three groups of people which the subject naturally suggests and the effects of such grouping upon the general subject. These groups are (*a*) Employers; (*b*) Employees; (*c*) The General Public.

To the employer of labor factory inspection has in many cases come to be looked upon with disfavor. Arbitrary regulations enforced by ignorant and tactless inspectors, the interference with one's private affairs, and additional expense are probably the most potent reasons for this state of affairs. I do not believe that the average employer objects to the establishment of reasonable sanitary regulations. He is too intelligent a man not to know that the employee whose environment is all that it should be in regard to pure air, proper temperature, proper light, proper toilet accommodations and freedom from exposure to contagious disease is of more value to him than the employee whose environment is unhealthy. I believe that the average employer, if approached in the right way, will prove a help rather than a hindrance to efficient factory inspection. In other words I think he needs education along health lines rather than additional legislation. Added to his interest as a manufacturer is his interest as a large tax payer in the general health of the community. In many cases the nature of the work done in the factory is such that under the most favorable conditions health is somewhat endangered; but I am inclined to believe that the so-called dangerous employments have been unduly advertised, and that inventive genius has, and will continue to minimize the evil. There are, however, certain obstacles to the production of ideal health conditions in thickly settled communities that are well nigh insuperable under present conditions. I refer

to the disposal of waste in communities where sewerage is not furnished by the municipality. My attention has recently been called to a case of this kind where an ideal handling of waste by the manufacturer would be so expensive as to be prohibitive. In such a case great care should be exercised to so handle the matter as to command the approval of intelligent public sentiment.

The employee is, of course, theoretically in favor of rigid factory inspection, and is, I think, as a rule willing to do his part to make it a success. Quite a large percentage, however, while apparently taking delight in seeing the employer compelled to spend money for sanitary appliances, are the first to abuse the facilities given them. Then, too, restrictions in cases of contagious disease are likely to prove unpopular. Here education along sanitary lines is also needed.

To the public factory inspection is of vital interest. The enormous expense which poor health brings upon the community, to say nothing of the humanitarian side of the question, should awaken the interest of every public spirited citizen in the subject. Then, too, the product of the factory has in so many cases a bearing upon the health of the consumer that the public cannot afford to allow it to be prepared under unhealthy conditions. I want to impress upon every medical man present the importance of the subject, and to suggest that you can by your personal efforts do a great deal to popularize and make effective scientific factory inspection. The medical man above all others is in a position to educate public sentiment along such lines. If the Commonwealth is to inaugurate an efficient factory inspection it must have an enlightened public sentiment in favor of such inspection.

In the crusade against tuberculosis now so well under way in the State, intelligent factory inspection is an important factor. The State has just made a large appropriation

for hospitals for the treatment of tubercular patients. Let us hope that in another decade we shall have so lessened the number of those thus afflicted that the hospitals will be poorly patronized.

It is interesting in this connection to note the work that is being done outside the State along the same lines. Pennsylvania has just appropriated one million dollars with which to fight the great white plague, and even our diminutive neighbor, Rhode Island, comes bravely to the front with an appropriation of three quarters of a million. With the work done in Germany recently you are all doubtless familiar. There seems to be a general awakening all along the line, and it is for us, the medical men of the old Bay State, to see to it that our beloved Commonwealth is in the very forefront of the battle.

DISCUSSION.

The Chairman, DR. A. T. CABOT, of Boston: The subject of factory inspection is now before us for discussion and is one which we should all consider carefully on account of its effect in prevention of tuberculosis. We should urge adequate factory inspection and try to enlist the help of employers to obtain it. Rhode Island has taken steps toward factory inspection which I think we might study with interest and profit. In their experience with factory inspection they have found that one of the most satisfactory inspectors is the foreman who is always in the work-room with the hands. He does not know all the symptoms of consumption perhaps, but he knows a cough when he hears it, and after hearing a man cough day after day, he is able to advise such a man to go to the doctor for examination. It is the cases which are early discovered which can be sent to a sanitarium. Those are the cases which come back to the factories, and are living examples to the workmen of the value to them of factory inspection.

DR. C. S. MILLET, of Brockton: In the city of Brockton during the past six months I have been trying to carry out this line of work in the so-called Walk-Over Shoe Factory, and all parties concerned,—that is, the members of the firm, the superintendents of the different factories and the employees,—have been willing and even anxious to aid me in every way.

In talking to the foremen, I have tried to tell them what to look for in their efforts to determine whether or not a man is probably affected and should have his lungs examined. The objective symptoms which seem to be most frequently the result of a tuberculous infection are four in number, and named in the order of their importance are: hæmorrhage, cough, emaciation and pallor.

First; Hæmorrhage:—Any spitting of blood, no matter how slight, should always be sufficient grounds for a careful physical examination. Persons who raise blood are quite apt to conceal it if possible, probably because of the curiosity and excitement which the sight of blood always arouses; hence, the foremen should always follow up the scent if they perceive any blood upon the floor or anywhere about the factory.

Second; Cough:—Osler has said that a cough which lasts over three weeks is very suspicious; and certainly if this symptom, even though it may be insignificant in character, shows the least tendency towards chronicity, the origin must be determined.

Third; Emaciation:—If a man is thinner than he should be compared with his height, or if he has recently lost weight to an appreciable degree without apparent cause, then again it is of the greatest importance to discover whether or not he has tuberculosis.

Fourth; Pallor:—This symptom one should always consider as at any rate suggestive enough to demand an explanation. It may be due to an actual anæmia, as shown by the test for hæmoglobin, or it may be caused by an unnatural condition of the circulation through the skin; and in either case the cause is so often tuberculosis that a physician must be absolutely certain as to its source before he can exclude the disease in question.

In conclusion, I would like to say that this work of seeking out cases of tuberculosis among mill and factory

operatives must always be done with caution and sometimes even with secrecy, in order not to make it possible for the other hands to shun or ridicule a fellow-workman who may have been subjected to an examination.

DR. W. J. SLEEPER, of Westford: I think that factory inspection will do a great deal to blot out the great white plague in that it will call attention early to any trouble in the chest, and I think also that it is very important to secure the coöperation of the employee.

The increase in speed in the factories, while it produces an increase of production, is certainly a detriment to the employee, and I see no way of correcting the condition except by means of the two weeks' vacation in the summer time. It seems to me that the giving of vacations for the purpose of recreation and change of scene, should be encouraged by our society.

DR. ELEANOR WAY-ALLEN, of Walpole: In Walpole a committee has been formed for the relief and control of tuberculosis, and we have found that people coöperate with us very well. We have put up "No Spitting" signs and are recommending men and women to physicians if we find they are failing in health. The largest employer has a physician at his own expense to examine patients. We have found that working with the foremen is a very successful method.

DR. J. C. PITTA, of New Bedford: In New Bedford, as you all know, there are many large factories, and they are a long way from the most progressive methods of fighting this disease. There is not a great deal of inspection. A large number of the people come from my country (Portugal). Last year there were over 120 sent back because they had tuberculosis. I tried to organize a society for the protection of our people, and we have succeeded in doing something. Some of them have been sent to the State Sanitarium at Rutland, and the bills paid by our Society. We have a nurse who goes around and takes their temperatures and give them good advice. We think we have done much work, but could do more if we had the assistance of other societies.

I am also a member of the District Committee in New Bedford, and for quite a number of months we have not had a meeting, due mostly to the fact that the president of the committee was sick, and afterwards was so busy that he could not give the time to it, and we got somewhat discouraged. Still we have done quite a good work after all. We succeeded in getting a tuberculosis exhibit in City Hall, and it was attended by quite a large number of people of the community. Most of the patients sent back to the old country, and most of the patients attended by this society have been mill hands. Many of them drink from the same cup, and they are constantly in a dusty and over heated atmosphere.

ARTICLE XXV.

LEAD POISONING IN A RURAL COMMUNITY.

By WILLIAM N. COWLES, M.D.
OF AYER.

READ JUNE 12, 1907.

LEAD POISONING IN A RURAL COMMUNITY.

THE purpose of this paper is to give an account of my experience with lead poisoning among the people of several country towns in my vicinity, viz: Harvard, Shirley, Littleton, Acton and Ashby, Massachusetts.

The persons affected have been chiefly residents of farm houses, using water drawn from wells or springs through lead pipe. I have observed the cases during the last ten years, at first rarely, but later with increasing frequency, as my interest in the subject and ability to recognize the condition in its various forms have increased.

No attempt will be made to treat of lead poisoning in general. The cases will be related as briefly and with as little detail as possible, consistent with establishing the nature of the affection, and the extent and variety of the damage due to this intoxication.

Mrs. W., 66 years of age, consulted me in June, 1899, on account of what she called rheumatism; she was well and active until her present illness. The trouble began eight months before, in the hands, soon spreading to the shoulders, neck, feet, and knee and hip joints, becoming all the time steadily worse.

The finger and wrist joints were swollen and stiff; there were pain and tenderness in the course of several large nerve trunks, both brachial regions being affected.

The pain was much worse at night; she walked with difficulty on the level, and could not ascend steps without help, she could not dress her hair, and fed herself with difficulty. There was no apparent paralysis. Lead poison-

ing was suspected, but inquiry showed that the water supply at her house was drawn from a well by means of a well-sweep and wooden bucket.

No source of lead could be discovered after persistent inquiry, until the patient recalled that about two years before an old lead clock-weight had been accidently dropped into the well. The well was pumped free of water, and the clock-weight found and removed. In two weeks Mrs. W. noticed an improvement in her lameness, and in four months she was entirely well. She lives at the same place and uses water from the same well, but has had no return of the lameness.

This piece of lead has about 32 square in. of surface. The smaller sizes of lead pipe have more surface than this for each foot of length.

Lead pipe in a well usually goes several feet under water, giving many times the surface of this weight for solution of lead from the *outside* of the pipe.

G. H. C., 46 years of age, lived with his family on a farm in New Hampshire, for twelve years, from 1891 to 1903, using spring water carried by lead pipe. In April, 1903, he moved to a farm in Littleton, Mass., where he also used spring water running through lead.

In the fall of 1904, a year and a half after moving to Littleton, C. began to be troubled with shortness of breath on exertion. His heart was enlarged, and his radial and temporal arteries tortuous and thickened. A year later lead poisoning was suspected; lead was found in the water used by the family, and iron pipe was put down in place of the lead. Seven months after the lead pipe was removed C. was given a course of potassium iodide, and lead was found in his urine.

At present he bears exertion a little better, but his heart is still enlarged, and his arteries still thick and hard. He has never before had any serious sickness, and comes of a long-lived family.

Mrs. C., 37 years of age, wife of G. H. C., a pale, thin, and delicate looking woman, after living a year and a half in Littleton, began to be troubled by obstinate constipation; the bowels could be moved only by powerful cathartics or enemata; the colon could be felt through the thin

abdominal wall like a solid cord. At times the introduction of an enema was impossible, except under the influence of a dose of morphine. The evacuation at such times consisted of small, hard lumps of feces, with much mucus in shreds and strings.

I made a diagnosis of mucous colic, and prescribed a coarse vegetable diet, as recommended by Von Noorden. The patient held to this diet faithfully for several months, with great relief. At this time I flattered myself upon the acuteness of my diagnosis, and the successful treatment of the case.

A year later, in the fall of 1905, Mrs. C.'s abdominal trouble returned, with great severity. There were intense colic, constipation, slow pulse, flat abdomen, a distinct lead line on the gums and the discovery of lead in the household water supply. The mucous colic had become plain lead colic; the usual treatment of lead poisoning brought complete relief; Mrs. C. is now well with gain of flesh and ruddy color, and has had no return of the colic either mucous or lead.

Mrs. G., mother of Mrs. G. H. C., suffered from a persistent and debilitating diarrhœa, for two and a half years from the time of moving to Littleton with her daughter, until lead was eliminated from the family water supply, when she recovered, and has been free from diarrhœa for the last year and a half.

T. G., 78 years of age, has lived in a farm house in Harvard, Mass., since 1899. The house was then supplied by water drawn from a well through 275 feet of lead pipe. Two years later, in the spring of 1901, his right hand, right leg, left hand and left leg, in the order named, became weakened to such an extent that he was confined to his bed. At the same time he suffered severe but intermittent pain. The abdominal symptoms and the weakness of his arms and legs became steadily worse, until September, four months in all, when the extensor muscles of both hands and both feet were paralyzed. His abdomen was hard and tender. There was a prominent lead line on his gums. The family was then supplied with water known to be free from lead. He made a tedious and partial recovery. At present, six years after his lead poisoning,

his grasps are good, he opens his hands partially and feebly only. He walks with some difficulty, and falls if his knees happen to become much bent. The extensor muscles of his forearms, thighs and legs are wasted and weak. He says he feels well. He has a ruddy complexion, soft peripheral arteries and a normal urine.

J. M., 47 years of age, has lived for the last ten years on his farm near West Acton, Mass. Seven years ago he had a serious sickness accompanied by great pain in the abdomen, for which he was treated at the Massachusetts General Hospital, where he was told that lead poisoning was the cause of his trouble. He has had a similar sickness every summer since.

I was first called to him in the summer of 1905, for a well marked lead colic, in which he had great pain, constipation, slow pulse, with wakeful and delirious nights. His skin was of a muddy pallor, his hair very gray, his radial and temporal arteries tortuous and hard. There was a plain lead line on his gums.

The family was supplied with water brought from a spring in milk cans. Projecting into this spring was a piece of lead pipe, once used for pumping water into the house. A little probing with a slender stick showed that the water entering the spring, came in alongside the lead pipe, after flowing a considerable but uncertain distance in contact with it. There was ample chance for solution of lead from the outside of an old pipe. J. M. was told that this lead pipe was the cause of his sickness, and he was advised to remove it.

A year later, in the summer of 1906, I was called to him again, because of a repetition of his colic, and found the lead pipe still in the spring. He was advised again to take it out. About a month after, he was attacked once more with colic, and the pipe had not been removed. I then told him that he must put away his lead pipe or get another doctor, and he finally got rid of the pipe. Lead was found in his urine at this time. His health has improved, but he is still far from well. After ten years of lead poisoning, at the age of 47 years, he looks, talks, and acts like an old man.

Mrs. P., 84 years of age, has lived for about fifty years in a house in Shirley, Mass. For many years (forty or more) she has been partly crippled with what she called rheumatism. She was not able to walk outside of her house for years on account of lameness of her knee, ankle and toe joints. Her hands were nearly useless. The wrist and knuckle joints especially were swollen and deformed. All these joints were painful and tender, the pain being much worse at night. Many kinds of treatment had been tried without benefit, several at my suggestion. The water supply at this house has always been spring water pumped a long distance through lead pipe. It has been examined and lead was found in it. Two years ago, at my suggestion, water was taken from the public supply through iron pipe, and the spring water was stopped. Since the change, and with no other treatment, Mrs. P. has lost her rheumatism entirely. She walks with ease and comfort. Her wrist and finger joints are free from swelling and she uses them with much facility.

Mrs. J. E., 64 years of age, consulted me in 1897 for "sciatica." There was also a history of rheumatism affecting the joints of the hands, with a long continued painful affection of one shoulder and arm, the pain being worse at night.

The water supply of the family was drawn through many rods of lead pipe. Lead was found in the water and in the patient's urine. Iron pipe was substituted for lead. Mrs. E. recovered completely and has been free from rheumatism for the last ten years.

Mrs. M. has lived on her farm in Harvard, Mass., 47 years. Water for her house comes from a spring, through lead pipe, and contains a dangerous quantity of lead. She has suffered for many years with what she calls rheumatism. Her finger and wrist joints are enlarged and distorted. She suffers pain and lameness in all her extremities. Her health in other respects seems good. Her color is good.

Nellie M., 43 years of age, daughter of Mrs. M., has always lived at home. She is pale and cachectic in appearence, looking much older than her years. She began to have pains in the extremities, which she calls rheumatism, about twenty years ago. Her left leg and thigh were first

affected. Lameness was troublesome, and pain was severe at times. For the last seven years she has suffered much pain in the left shoulder, arm, forearm and hand, worse at night. The left hand trembles with a moderately coarse tremor. She suffers from frequent and severe headaches. These people have been warned many times in the last few years that their water supply is dangerous, but they continue to drink the lead water and suffer the pains.

W. K., 57 years of age, has lived on his farm in Harvard, Mass., eighteen years. During the last five years he has had sciatica, brachial neuritis, and many other painful ailments of an indefinite nature. In March of this year he was seized with severe headache which lasted for two weeks. He has the appearence of a very old man; his heart is enlarged; his radial and temporal arteries are thickened and hard. His house is supplied with water from a spring, through lead pipe. The pipe corrodes so much that it leaks and requires repair several times each year. The water at the house contains considerable lead. K.'s urine contains a large trace of lead.

Mrs. K., 51 years of age, wife of W. K., looks much older. Her hair is completely white. Her skin is of a grayish pallor.

In the Spring of 1906 she had a severe brachial neuritis. A year later she suffered from a sharp attack of lead colic. K. had been warned of the danger of lead poisoning by Dr. Royal of Harvard and myself many times during the last few years, but he has only lately become a believer and turned from drinking lead water.

A. S., 54 years of age, first consulted me in 1904, on account of a troublesome vertigo. His heart was enlarged; his peripheral arteries thickened and tortuous; he had a pulse of high tension and the urine of chronic interstitial nehpritis. Lead was found in his urine. For ten years he had been using rain water stored in a cistern with distributing pipes of lead. A year later, in 1905, a progressive cerebral degeneration appeared in S., and he died in the summer of 1906.

Mrs. S., Harvard, Mass., in middle life, consulted me several years ago because of recurring pain in the abdomen.

She had a plain blue line on the gums and was using water brought to her house through lead pipe. I warned Mrs. S. and her husband of the danger of using the contaminated water, but they were skeptical and clung to the lead pipe. Dr. Royal, of Harvard, tells me that Mrs. S. has suffered several attacks of brachial neuritis in the last few years, and that he has had the water at her house examined for lead, with a positive result. In spite of these warnings they refuse to abandon the lead pipe.

Mrs. R., 46 years of age, has lived for ten years in a tenement house in Shirley, Mass. She suffers greatly with neuralgic attacks, affecting at various times the head, face, both brachial regions and the right crural region. Such treatment as she has received has been of little use. Her house is supplied with spring water carried in lead pipe. Examination of this water for lead is positive.

Mrs. C., 70 years of age, has lived in a cottage house in Harvard, Mass., since 1891. During much of that time she has been greatly troubled with "rheumatism." In the autumn of 1903 she had several attacks of angina pectoris. She was very pale, with swollen ankles and the urine of a chronic nephritis of moderate degree. The family used water drawn from a well through lead pipe. Examination of the water for lead gave a positive result, and its use was stopped. Mrs. C. passed a poor winter with much cardiac distress and œdema of the legs. In the Spring of 1904 she got some relief and has gradually improved since. She now has the urine of chronic interstitial nehpritis, with a pulse of high tension and good cardiac compensation. Her life is tolerable.

George H. C., 70 years of age, husband of Mrs. C., lived in the house now occupied by them from 1850 to 1863, and from 1891 to the present time. He has been the victim of dreadful headaches for many years (40 or more). At the time of the discovery of lead in C.'s well water (1903), he was pale and emaciated and suffered frequently from severe headaches, lasting several days at a time. Since the lead water was stopped he has improved greatly in health. He has lost his headaches, has a ruddy color and has put on much flesh.

From 1894 to 1903 Mr. and Mrs. C. passed several winters in California, being much pleased with the effect of the change of climate upon themselves, he always losing his neuralgia and she her rheumatism in California, only to find them again in Massachusetts. Since stopping the lead water they have been as free from these ills in Massachusetts as in Calfornia.

Mrs. C., mother of G. H. C., lived in the same house, with the same water supply, from 1850 until her death in 1880 at the age of 82 years. In 1855 she began to be troubled with "rheumatism," which gradually increased, affecting the hands and feet chiefly. She was completely crippled for the last thirteen years of her life, having comfortable days and intolerable nights.

CONCLUSION.

This group of cases is but another illustration of the many forms in which lead poisoning makes its appearance, and the various kinds of damage done by it.

The acute cases are few; the chronic painful and disabling affections and arterio-sclerosis, with the things that go in its train, are in the majority.

My own experience is one of failure for years to recognize lead poisoning as a frequent cause of many of the painful and disabling ills that I have been called upon to treat. I recall many instances of vague abdominal troubles, and of painful and crippling affections of the extremities which the patient called rheumatism, and for which I have no better name, that I tried vainly to relieve, and which may well have been due to poisoning by lead.

Other physicians may have failed to recognize lead poisoning as I have done. Many people look upon what they call rheumatism as something to be endured without hope of relief, and the cause of their ailment is never discovered. Arterio-sclerosis comes on insidiously, until some terminal accident occurs, and lead as a possible cause is rarely thought of. It seems fair to assume therefore,

that my cases are but a fraction of those occurring in the same locality, the whole making a heavy burden of pain and disability upon the community.

The use of lead pipe for carrying water is common throughout the whole of rural New England. The mischievous quality of dissolving lead differs, no doubt, in the localities and at different times. But I believe such use of lead is always and everywhere dangerous, and that the possibility of serious poisoning ought to be more generally recognized than it now is.

DISCUSSION.

DR. F. C. SHATTUCK, of Boston: This paper of Dr. Cowles' seems to me very important and very interesting. It is especially important, it seems to me, to those practitioners who reside in the country, in rather thickly settled regions where there is not a public water supply. I do not mean to say that we do not encounter cases of lead poisoning here in Boston and in other cities; we do, but such an experience as Dr. Cowles' is remarkable to me, and I am sure it cannot be an isolated one.

There are two or three points on which I would like to speak. In the first place I think that one of these cases which Dr. Cowles mentioned as being brought to the Massachusetts General Hospital, and where a diagnosis of lead poisoning was made, if I remember correctly, was sent in on the surgical side and came near being operated on for appendicitis. In this hospital there are many cases of bellyache sent in for immediate operation, and they have had the same experience at the City Hospital. The appendix is not the only thing which may produce pain.

Another point is the matter of individual susceptibility to lead poisoning, which I think is very striking. Dr. Cowles seems to dwell upon it, so I am sure he has been struck by it in his experience. Of members of the same family, drinking the same water, exposed to the same danger, some suffer and some do not. In other words, there are some

people who have a far greater susceptibility to lead poisoning than others. I have in mind a case of a young girl at the Massachusetts General Hospital. She was one of a family of nine living in Blackstone and working in a mill. She had been ailing, and it was thought that she was tired and overworked and needed rest, so she went away from home just as one of Dr. Cowles' patients went to California. She made a visit in Dorchester, and the air, etc., of Dorchester agreed with her very well. Then she went home and got sick again. Out of the family of nine only she and her mother had symptoms, and she had not drunk so much water as many other members of the family, because she took her dinner with her to the mills, and had only two meals at home, while all the others had three.

There is another point which was enforced upon me by the paper; this is the obstinacy exhibited by some of our patients. Dr. Cowles has read to us three or four cases where, although the people have been warned once, twice and even three times that the source of their trouble lay in a bit of lead about their wells, they would not take the trouble to get it out, even on the chances.

Dr. F. J. Canedy, of Shelburne Falls: I practice in a country region largely, living in a village of 2500 inhabitants, and I also have many consultations with neighboring physicians. Although probably 95 per cent. of these people have water supplied from lead pipes, as is the case in most country places, the cases of real lead poisoning are quite rare. While I must plead ignorance concerning some of the cases, my experience has been that in most instances of this kind, some cause other than lead poisoning has been found. I have several times suspected some of my cases and have had the water examined, and have never found anything the matter with the water conducted through lead pipes. Indeed it is the experience of the farmers who have iron pipes that the small pipe which carries the water becomes so rusty that it is no longer useful, and for this reason most of the iron pipes in my region are being taken up and lead pipes substituted.

If it is a fact that there is so much danger from this source, it ought to be very generally known and thoroughly disseminated.

DR. E. B. HARVEY, of Westboro': Will Dr. Canedy state to us the character of the water in his region? Is it soft water, or is it water containing a considerable quantity of lime?

DR. CANEDY: It is not hard water which precipitates quantities of lime; neither is it very soft water.

The President, DR. GAY: On a recent visit to the New Hampshire Medical Society this subject was brought up with experience very much like Dr. Cowles'. One man had had 30 cases, and others reported a great number.

It was a revelation to the Chairman that it was so common in the country districts, as we have so little of it in this city.

ARTICLE XXVI.

STENOSIS OF THE PYLORUS IN INFANCY.

By CHARLES L. SCUDDER, M.D.
OF BOSTON.

READ JUNE 11, 1907.

STENOSIS OF THE PYLORUS IN INFANCY.

We all are more or less familiar with stenosis of the pylorus in adult life, whether it be caused by a benign or a malignant lesion. Stenosis of the pylorus occurs also in young babies. It presents a unique pathology — benign in its anatomical characters but most malignant in its fatality. It occurs with greater frequency than is generally supposed. It is too often overlooked. The diagnosis is sometimes confusing. One type of the disease is almost invariably followed by death if treated unintelligently.

It is for these reasons that I propose to present for your consideration the salient facts concerning stenosis of the pylorus in infancy and its surgical treatment. These are subjects of the very greatest practical importance to the general practitioner of medicine, the pediatrist and the surgeon.

Frequency.

Scudder and Quinby, in May, 1905,* two years ago, analyzed all cases of this disease. During 17 years literature, since 1888, 115 cases were recorded, 55 autopsies and 60 operations. Since this time, during the past two years, from May 1905 to May 1907, about 40 cases have been reported. Papers upon this subject have contained the records of isolated cases treated medically and surgically. No very important addition to our knowledge of the subject has been made during this time.

* The Journal of the American Medical Association, May, 1905.

The pathologist at autopsy and the surgeon at operations upon the living baby have together convinced all hitherto doubting pediatrists and internists that this disease exists as a distinct entity and that it demands active consideration in the differential diagnosis of the acute abdominal diseases of early life. In those communities in which there exists an accurate knowledge of this disease cases are being repeatedly discovered. Stenosis of the infantile pylorus is not uncommon.

Pathology.

The findings are constant and uniform. Occupying the position of the normal pylorus is a tumor, small, smooth, oval, hard, movable and without adhesions. The tumor varies in size, averaging that of a hickory nut. This tumor has a constant structure — an hyperplasia of the circular muscular fibres of the pyloric segment. There is also either an hypertrophied or a thinned stomach wall, occasionally a dilated œsophagus, a narrowing of the pyloric lumen, an hypertrophy of the mucus membrane and a collapsed empty intestine.

Etiology.

The cause of the hyperplasia of the circular muscular fibres is unknown. There are three hypotheses suggested to explain the conditions: —

1. That of Pfoundler — a spasmodic contraction due to post natal gastric irritation.
2. That of Nicoll — a simple overgrowth of the sphincter muscle of the pylorus.
3. That of Thompson — an incoördinated action of the stomach muscles dependent on functional disorders of the gastric nervous system, resulting in gastric spasm.

A discussion of the etiology is fascinating but unprofitable.

Clinical Picture.

An apparently healthy breast-fed child is born. Upon the third or fourth day he vomits whatever he may have

taken from the breast. The vomiting continues at irregular intervals, and is evidently independent of the quality of the food. The child loses weight. All kinds of artificial foods and modifications (home and laboratory) of cow's milk are used without permanent success. He wastes away, weakens and dies.

The cause of death affixed to the certificate is one of the following: "Infantile atrophy," "Gastric irritation," "Sub-Acute gastritis," "Inanition," "Chronic gastritis," "Gastro-intestinal catarrh."

If in cases of this kind observations were made with accuracy the following significant facts would often be found: — the baby's tongue is perfectly clean; the breath is sweet and natural; the appetite is a little less ravenous than usual; though hungry he does n't feed greedily, he is soon satisfied.

After feeding, the baby shows signs of abdominal discomfort, his face is covered with grimaces, he wriggles about uneasily, he even cries with evident pain, he then vomits. The vomiting is very forcible, through the nose as well as mouth; is often mistaken for a simple regurgitation from a full stomach. The hypertrophied stomach wall contracting violently against the obstruction at the pylorus, causes the gastric contents to be expelled forcibly through the relaxed cardiac orifice.

The frequency of the vomiting is dependent upon the number of feedings, and upon the amount taken at a feeding. The quantity vomited varies with that received into the stomach. The material ejected is the milk and food taken. There is usually no bile, no mucus and no blood in the vomited material. There is almost never hyperacidity of the stomach contents. After the vomiting the baby is quiet and sleeps until he next feels hungry.

If the abdomen is carefully palpated a distinct tumor may be felt above and to the right of the umbilicus. This

tumor being small and partly covered by the liver is not always readily felt. An enlarged mesenteric gland has been mistaken for a pyloric tumor. If the abdomen is inspected in a good light immediately after a feeding, strong gastric peristalsis will be noticed. This was very evident in two of the cases I have to record.

The dejections are somewhat variable in character, but because of the slight amount passing through the pylorus they are small and meconium like, occasionally fecal in character, at times watery.

The stomach will be found dilated in proportion to the duration and degree of the obstruction. Epigastric fulness and a sunken lower abdomen are suggestive.

The child loses in weight despite treatment, because of starvation.

Diagnosis.

Characteristic vomiting; a pyloric tumor; visible gastric peristalsis; gradual and progressive loss in weight despite skilled feeding; this sequence of these signs is significant. An absence of a *seemingly important* sign is of comparatively little consequence. The total picture is most suggestive.

The obstruction to the pyloric lumen caused by the tumor is variable. Difficulty in diagnosis is measured by the degree of obstruction. There is little doubt about the diagnosis if the obstruction becomes complete. If on the other hand the obstruction is incomplete or partial, there will be doubt.

An obstruction may result from a tumor at the pylorus, also from a spasm of the pyloric muscle independently of tumor formation. The symptions may simulate a chronic gastric indigestion. Each of the signs found in cases of pyloric stenosis may be present in cases of infantile indigestion, but the particular quality and sequence of symptoms characteristic of pyloric stenosis are absent. The completed

picture is wanting. Vomiting is not always symptomatic of obstruction in young babies, cerebral disease may be imminent. There is little danger of confusing an intestinal indigestion with pyloric stenosis.

Pyloric stenosis in infants if untreated is usually fatal. A few cases are reported in which medical treatment is said to have effected a cure. Unfortunately no medically treated case has been demonstrated to have had the disease. Surgical treatment has cured more than half of these desperate cases.

Operative Treatment.

The degree of the obstruction and the general condition of the child should be the basis for treatment. The cases arrange themselves into three groups: —

1. The obstruction is evidently complete or almost complete — operation is demanded at once.
2. The obstruction is partial and the child is not doing well, and is losing weight — operation is likewise indicated.
3. The obstruction is partial and the child is doing fairly well, and is not rapidly losing weight. With this third group one may be justified in experimenting with medical treatment, but only for a short time. It must always be remembered that remissions of symptoms occur. Improvement may be but temporary, the strength of the child must not be allowed to greatly diminish before operation is done.

In this third group (in which obstruction is partial, and the child is doing pretty well under medical treatment) are found the so-called cases of "pyloric spasm" which are supposed to yield to medical coaxing. Such is the case reported by J. L. Morse in Sept., 1906.*

It is more and more evident that prolonged medical treatment of certain of these cases is irrational. The risk and danger to the child's life is often greater than if oper-

* Boston Medical and Surgical Journal, Sept. 27, 1906.

ative treatment were instituted early. Heubner of Berlin presents a series of cases all treated medically without operation. His mortality rate is low. I believe from a careful analysis of his cases, that deductions cannot be drawn from his twenty-one cases till they are followed a longer time. It is questionable if he had to with true pyloric stenosis.

There are very real dangers attending the treatment of this disease. The immediate danger is apparent, viz. the letting slip the opportune time for operation, misjudging the condition of the child. The later danger is that there may occur in youth and in adult life gastric difficulties dependent upon the persistence of infantile conditions. Instances are recorded by Beardsley, Sonnenberg, Hansy and Shaw which make this latter possibility very real. Both of these dangers are probably eliminated by operation.

The mortality following operation still remains high — but the mortality of operation upon weak and wasted infants will always be large. Many different operators with varied techinque and the consequent absence of exactness will always record a high mortality in these desperate cases. An exploratory laparotomy upon a young infant in which the diagnosis is in doubt, and in which improvement has ceased is certainly wise. I have to record one such exploration. The operation of choice in cases of pyloric stenosis is a posterior-gastroenterostomy — done with clamp and suture. The Loreta operation of divulsion and Pyloroplasty are two other procedures thought well of by some.

There is little doubt of the existence of cases which simulate pyloric stenosis with tumor, and which improve and apparently recover under some one or more of the following procedures: resting the stomach; lavage of the stomach with weak alkalis; warm abdominal poultices; nasal tube feeding; the adminstration of opium and atropine;

the giving of opium before feeding (one-eightieth of a grain); the use of alkalis; experimentation with food by changes both in quality and quantity; rectal feeding.

That there actually are cases* of pyloric stenosis with tumor, which have recovered completely under medical treatment, has not yet been demonstrated. Until this demonstration is made, I prefer the certainty of surgical treatment to the uncertainty and very high mortality of medical experimentation. Seefish in 1904 mentioned three cases which "recovered" under medical treatment, and were all operated upon before 12 years of age. Surgical supervision of medically treated cases will help to diminish the large mortality of cases treated medically.

From my own limited experience of the past two years, I have to report five cases. I will not at this time relate these cases in detail (two have been previously recorded), but will mention briefly certain facts of interest concerning each.

In the first case operative treatment was withheld because the child was moribund. The stomach of this child exhibited at autopsy a typical pyloric tumor and an hypertrophied stomach wall.

In the second case an exploratory operation was done upon a very sick child, and the tumor was not present. The child has since recovered, and is after a stormy experience with various foods increasing in weight.

Of the three remaining cases, in each of which a posterior gastro enterostomy was done:

Case 3† has the distinction of being the youngest case yet operated upon; he was fourteen days old. He had been artificially fed. The stomach was not very large.

* Such are supposed to be the cases recorded recently by Reinach, Marx, Murdock, Morse, Harper, Griffith, Sutherland, Hutchinson, Neild, Rob, Crahim, and Heubner, all carefully observed and each treated by medical means. The cases have been followed and are known to be free from all signs and symptoms of gastric disturbance from several months to two and one-half years after treatment ceased.

† Boston Medical and Surgical Journal, Dec. 14, 1905.

Case 4* was twenty-four days old. He had vomited from the third day after birth. The stomach wall was thickened. He had been breast fed.

Case 5 [recorded here for the first time; to be reported in full elsewhere] had likewise been breast fed. He was twenty-two days old. The stomach wall was edematous, and markedly thickened in the pyloric part.

In the last three cases the signs of the disease were typical. The tumor was characteristic. All three cases recovered from the operation. All have been followed, and each child is now robust and well, two years, one year and ten months, and seven months, respectively, since the operation.

Sufficient time has now elapsed to make it possible to know the real results of a gastroenterostomy in these cases. I have succeeded in determining the condition of certain cases of other observers several years after operation. The cases of Abel, Löbker, Fritzsche, Kehr, Schotten, Giles, and Stiles quoted in the original paper,† are alive and well; four of them six years, one four years, one three years, and one two years following operation. These results are significant. They demonstrate: 1, that these presumably otherwise normal children have escaped the difficulties of obstruction; 2, that nutrition does not suffer following gastroenterostomy in cases of infantile pyloric stenosis. Posterior-gastroenterostomy is an effective measure under these conditions.

These cases of gastroenterostomy following pyloric stenosis in early life form a series of great experimental value. The operation is done under almost normal physiological conditions, excepting for the mechanical obstruction. Presumably no other disease exists in these tiny new born individuals. There is very little disturbance of digestion. The results are absolute. The effects produced by the operation are uninfluenced by disorders of digestion, and

* Boston Medical and Surgical Journal, Feb. 22, 1906.

† The Journal of the American Medical Association, May, 1905.

other diseased states so commonly found in adults. These results, therefore, go far to confirm and substantiate the evidence accumulating from adult cases that posterio-gastroenterostomy properly performed is physiologically harmless. Pyloroplasty and gastro-duodenostomy, whether of the Kocher or Finney type, are theoretically and physiologically ideal procedures. Posterio-gastroenterostomy is proved to be physiologically satisfactory, and in these cases of infantile stenosis is certainly anatomically the choice of all procedures.

I may be pardoned if I mention here some of the practical matters which one treating these cases surgically will find helpful.

Preparation of the baby for operation. The baby is weak. A nutrient and stimulating enema containing salt solution and whiskey, to make ʒ ij, given very slowly through a soft rubber catheter, will help the child remarkably, especially if there is time to repeat it before operation. The stomach need not be washed out, for the vomiting which has persisted has usually left the stomach empty. The skin of the abdomen is very sensitive. Local preparation should be restricted to bathing the abdomen after etherization, first with castile soap and water applied with a gauze sponge (green soap is to be avoided) and then with warm 70% alcohol. The arms and legs are to be covered with sheet wadding or an equivalent, secured best by a little bandaging. The baby may lie upon a blanket, underneath which are two or three warm water bottles, the water not being warmer than 100° F.

The *anesthetic* is perferably ether only. This should be administered by a most skilled anesthetist. Little ether is needed after the abdomen is opened. It is essential that the anesthetic be continued sufficiently to preclude the likelihood of a prolapse of gut from the wound. There is very great shock to a little baby from over etherization.

The *instruments* should be adapted in size to the very small parts to be manipulated. Small short-toothed forceps and small scissors should be used for work upon the gut. Retractors are not needed and their use should be avoided. Retractors may increase shock, traumatize the wound and stimulate post-operative adhesions. I believe that a small clamp is efficient in preventing hemorrhage from the bowel wall, in diminishing soiling of the peritoneum by intestinal contents, and that it facilitates the suture. A needle holder is unessential — it's a time consumer — the fingers make the best needle holder in the operation of gastroenterostomy. A zero or double zero chromic catgut is satisfactory for the inside intestinal suture, while a fine Pagenstecker linen thread is secure for the inside suture in the gastroenterostomy.

The Operation per se.

The *abdominal wall* is extremely thin. The abdomen may be opened unexpectedly. The *liver is large*. The median incision will be best made both above and below and to the left of the umbilicus. The incision should be large rather than small, perhaps three inches long. The small opening necessitates greater handling of the abdominal contents. The little larger opening enables the operator to secure the jejunum very readily without the trauma of much handling. Nothing should be removed outside the abdomen excepting the parts to be operated on, the stomach's posterior wall and the jejunum. The great omentum, containing little or no fat tissue, is drawn upward as a thin film from over the intestines. Care should be exercised lest the very transparent and veil-like thin gastro-colic omentum be incised too vigorously and the stomach wall itself suffer by being nicked by the knife. The *mesentery of the jejunum* is short in proportion to the width of the gut as compared with the adult mesentery, consequently it is with difficulty at times that the intestine can be brought forward to the

anterior stomach wall for an anterior gastroenterostomy without constricting the transverse colon unduly. In my cases I have made no loops, and have placed the stoma near the origin of the jejunum. The *stoma* in the infants' intestine should be from three-fourths to one inch long when sutured. If a very large stoma is made, obstructive difficulties may be caused by the wall of the gut opposite the stoma blocking its lumen, as suggested first by Cannon. This I have had happen following gastroenterostomy upon cats. After suture, the parts are returned to their natural places so far as possible, the jejunum to the left of the spine.

In *closing the abdominal wound* no attempt should be made to close by layers. Through and through sutures of silk worm gut are satisfactory. These sutures should not be drawn too tight, for the suture cuts its way through the young skin readily. Some one has suggested that these sutures be tied over bits of tiny rubber tubing to prevent them cutting the skin.

In placing the *sutures* the needle should avoid wounding the large vessels of the suspensory ligament of the liver which may lie very close to the right edge of the wound. The sutured wound is covered with a gauze sponge, and the abdomen is supported by two or three narrow straps of zinc oxide adhesive plaster. Traction on the sutures is reduced thus to a minimum. The position of these plasters may be changed to avoid their chafing the skin. An ordinary binder helps to support the abdomen.

After Operation.

Feeding the Baby. The importance of the proper feeding of these tiny little babies after operation for pyloric stenosis cannot be overstated. If the intestinal suture is well placed, the stoma is strong and is in less danger of leaking at first than after some ten days. It is common experience with suture of the intestine in the adult that the second week is the time of death from leakage. The feeding

should, if practicable, be in the hands of a skilled feeder of babies. Breast milk is the ideal food. Difficulties arise rapidly if an artificial food is needed. The amount to be allowed at each feed must be tentatively determined; fairly large amounts are usually well borne immediately after the operation. The baby is in great need of water, and it is a mistake to keep the total quantity low, especially if the food used agrees.

If breast feeding is possible the baby is allowed to nurse for two minutes at first. The time is gradually lengthened. If breast feeding is impossible it is best to begin with a whey, half ounce every two hours. Two days later may be used a weak modified milk, two per cent. fat, five per cent. sugar, five per cent. proteid, five per cent. lime water, and equal parts water. Gradually one may increase the richness of this mixture. Post operative rectal feeding may be needed for a few days. Salt solution enemata are helpful.

The *temperature* is often high even though there is no evident sepsis. In two of my cases the temperature immediately after operation was 104° F. and 105° F. It represented a normal reaction to this operation. This temperature gradually dropped during six days and then remained normal.

Even though the operation is done upon a weak baby it is not productive of great *shock*. The baby behaves very much in this particular as the adult after a stomach operation.

Vomiting does not cease with the operation. Vomiting persists for several days and ceases gradually. There was no vomiting in my first case (No. 3) after seven days; in my second case (No. 4) after six days; in my third case (No. 5) after about six days. This post-operative vomiting is less forcible than the vomiting previous to operation, and of course contains a little bile, and may contain a little old blood the first twenty-four hours. The post

operative vomiting may cause a breaking open of the wound, unless it is carefully sutured, with resulting prolapse of the intestine. This has happened in a few reported cases. In one case with which I am familiar replacement of the gut under ether was followed by a perfect recovery.

From the anterior abdominal wound may start an oozing of *blood* the second or third day. This occured in one of my cases (No. 5). It was difficult to control this oozing until a suture was taken through the whole abdominal wall. The amount of blood lost was not great, but was sufficient to make an appreciable difference in the facies of the child. Little babies bear a hemorrhage poorly.

There may be considerable *abdominal distension* following operation. This is relieved by small suds and glycerine enemata. In one case (No. 2) of exploratory laparotomy, six days after birth, there was considerable difficulty in keeping the sloughing *umbilical cord* from infecting the abdominal wound at the time of operation. Immediately after operation the cord was kept dry with boracic acid powder and neither a primary nor a secondary infection occurred. A few cases are recorded in which convulsions followed operation. These convulsive movements may be allied to the cases of gastric tetany occurring in adults. One case with which I am familiar recovered after these convulsive attacks.

Hernia, or a bulging of the abdominal wall at and in the region of the cicatrix, has occurred in two of my cases. This bulging has not proved sufficiently great to demand operation. A snug abdominal binder should be worn for one year after operation. The suture had best be removed on about the seventh day, or before much cutting of the skin is present, and the abdominal wall supported by zinc oxide adhesive plaster strips. In no group of cases with which I am familiar is the elimination of all the causative factors of shock so rapidly effective in establishing convalescence as in these cases of infantile pyloric stenosis.

Speed, implying familiarity with one's technique; absolute asepsis; hemostasis; a minimum amount of handling of the gut; an absence of trauma to the abdominal wound; conserving the body heat—these are the requisites of success in operations upon these tiny individuals.

DISCUSSION.

Dr. J. C. MONRO, of Boston: Dr. Scudder lays stress upon the presence of a tumor. We have had four of this class of cases, and in one of them which recovered there was no tumor to be felt, even under ether, before the abdomen was opened.

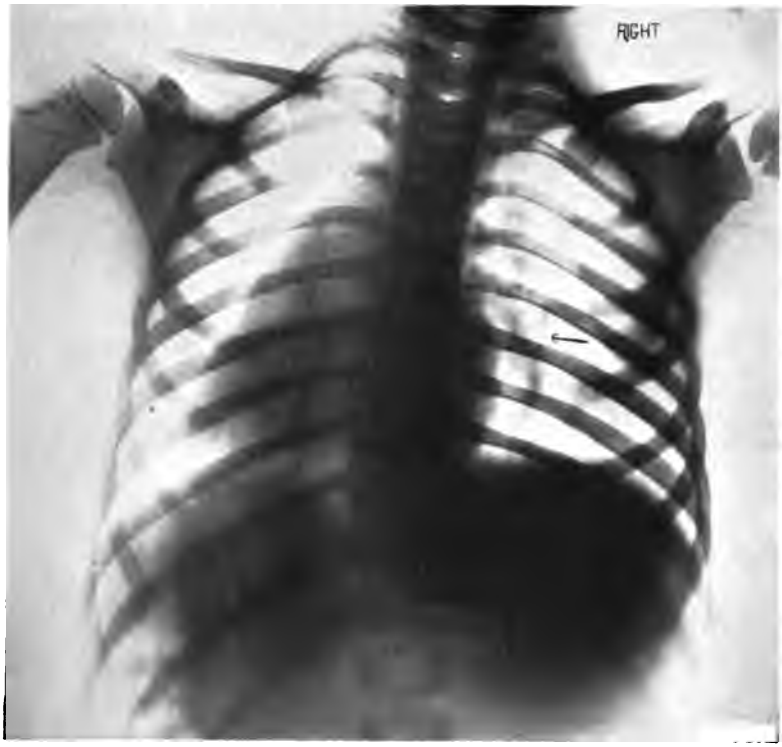
Another point that we learned from our cases is the importance of after-feeding. These cases, like almost any case of persistent obstruction of the pylorus, are apt to have dietetic disturbances after operation because of the unaccustomed presence of food in the intestines. We have had but one permanent recovery out of four, and in that case we found that the after-feeding was as important as the operation itself.

ARTICLE XXVII.

THE ROENTGEN RAY IN PEDIATRICS.

By THOMAS M. ROTCH, M.D.
OF BOSTON.

READ JUNE 12, 1907.



CASE I. PLATE I. (A. W. GEORGE, M.D.)

Case of enlarged bronchial glands, girl of 12 years. Notice dark shadows situated along right border of heart, extending up into apex. Seen in all normal chests, but to less extent than this. Lungs and pleura otherwise normal.

CASE II. PLATE II. (A. W. GEORGE, M.D.)

Case of foreign body, nail, in lower lobe of right lung, boy of twelve years. Nail inhaled five years ago and not suspected then, as case was examined by Roentgen method, and foreign body not recognized. Treated since for various diseases of lungs and pleura. Roentgenograph shows nail surrounded by a dark shadow, indicating thickened pleura and collapsed lung. Operation not successful.

THE ROENTGEN RAY IN PEDIATRICS.

ALTHOUGH the use of the Roentgen method has for some time been recognized as an important addition to our knowledge of disease, especially of the bones, yet its use in our routine work among children has been comparatively limited, especially in the so-called medical cases.

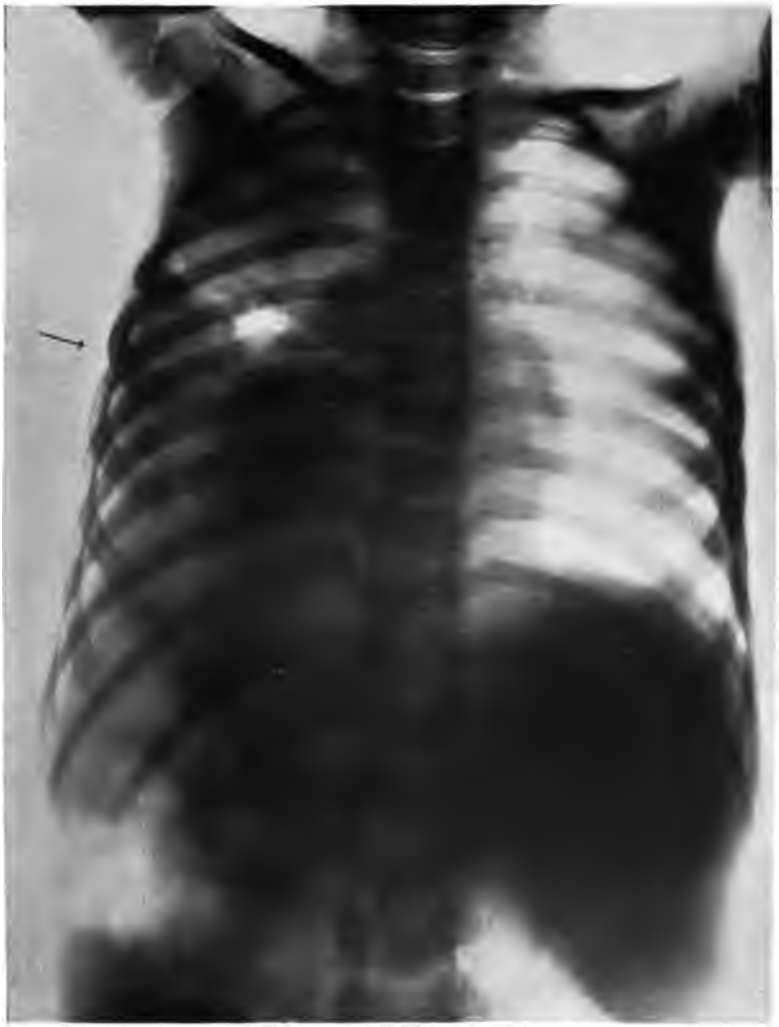
The value of the Roentgen method in children can best be tested by a systematic study of cases in the wards of a children's hospital, where each case can be kept under observation continually from the beginning to the end of the disease, and where all the other methods of diagnosis and treatment are at our command, in the hands of experts connected with the Hospital equipment. In a hospital also there arises no question of expense to the patient or consent of the parents. The hospital physician is free to use his wards and the hospital facilities as a clinical laboratory, unbiased by anything but the good of the patient. Such a clinical laboratory, thoroughly equipped through the wisdom, liberality and humane foresight for accomplishing the greatest amount of benefit for the patients, has been placed at the command of the physicians and surgeons connected with the Children's Hospital by the Managers, and it is my good fortune to have had rare opportunities for expert study in the medical wards under my charge. This opportunity has been greatly enhanced in value by the generous aid given me by my colleagues on both the surgical and the medical sides of the Hospital: aid which can only be appreciated when we consider how closely the diseases of young children are interwoven in medicine and in sur-

gery. Beginning, perhaps, as a surgical trauma and ending with a general septicæmia. Beginning with a tubercular infection of the hip joint and ending with terminal infection of the meninges. Beginning with a pneumococcus infection of the lung and ending with an infection of the pleura or peritoneum. Beginning with obscure pains in the joints, which would naturally make the parents think of rheumatism and so send for a physician, and ending with a rapidly developing osteomyelitis rendering operative interference imperative. Beginning, again, with a subperiosteal hemorrhage, with symptoms such as would lead to surgical advice, and before the true scorbutic and medical aspect of the case has been recognized, terminating in destruction of bone and surrounding tissue.

For the complete study, then, of an individual child, a combination of expert medical, surgical, pathological, bacteriological and chemical knowledge is required, with all that a hospital gives in expert work on the blood, the spinal fluid, the fæces, the urine, and, in young children most important of all, the ear and naso-pharynx.

As one of the primary aids to diagnosis and often alone determining the treatment in both medical and surgical cases, the Roentgen method of examination is rapidly taking its place, not only in rank with all the others, but in many cases proving the expert physical examinations to be wrong, and influencing the resulting lines of treatment to so great a degree that to diagnosticate and treat without this proof of the correctness of the diagnosis has in many cases proved to be foolhardy, dangerous and without excuse.

The knowledge obtained from the Roentgen ray, although at present confined to certain specific conditions, is continually widening, and it is well for the medical profession to recognize this fact and to assist in still further establishing its position among the instruments of precision to aid us, and in encouraging the perfection of its technique.



CASE III. PLATE III. (A. W. GEORGE, M.D.)

Case of plastic pleurisy in girl 6 years of age. Shadow extending over entire left chest excepting in central part. Shadow not changed with position.

CASE IV. PLATE IV. (A. W. GEORGE, M.D.)
Case of acute miliary tuberculosis in girl of 12 years.

There are three chief objects to be borne in view if we are to make a systematic study of the Roentgen method among children. First, the technique should be carried out by experts who have been thoroughly trained for this work, and in whose hands a failure to make a successful plate is reduced to a minimum. The reason for this is that children are so restless and difficult to take that it is much more important than in adults to have the first plate successful. Experts, also, who understand the temperaments of children and possess the faculty of managing them quickly and successfully. This necessity can only be appreciated by watching the work as it is done in the Roentgenological Laboratories of a children's hospital. Experts from long experience can make their work uniform by placing the tube at a uniform standard distance from the object taken, and the object in a uniform position and standard angle. This is especially necessary when we come to compare the normal anatomy of the part taken with disease of the same part, and emphasizes the fact that the use of the Roentgen apparatus in the hands of the practitioner in general almost invariably ends in imperfect and inexact results, and results which tend to throw discredit on the Roentgen method.

Second in importance is the full, intelligent and expert interpretation of the plate. Although this seems at first sight so simple, so easy to accomplish: — namely, to look at a picture and see what one sees — yet in the present state of our knowledge the result, when a number of physicians are examining a plate, is liable to be as many opinions, as so often happens when a number of opinions are given on an obscure cardiac murmur. Even the expert has not begun to learn what can be seen and will be seen when the interpretation of plates in the future has been further developed by broader experience and by careful comparison with post-mortem findings. For the picture is there, and all that we have to learn is to determine the language and

learn to read it. I have, for instance, had some Roentgenographs taken to determine the presence or absence of an infantile pyloric stenosis, and yet expert knowledge has failed as yet to read these plates, although the picture is directly and plainly before our eyes. We should then appreciate of what vast importance for future diagnosis is the perfection of interpretation. This can, of course, only be accomplished by those who are continually working, studying and devoting many hours a day to this work in a children's hospital, where a large number of cases can be kept under daily observation, and the clinical course and results of an especial case compared with plates showing the progress of the disease. Great progress can be made in this way in the interpretation of pleural and pericardial effusions, pulmonary solidifications and the rapid changes which are so common in the degrees of cardiac dilatation in young children.

So much impressed have I been, when following up my clinical examination, with the ever increasing expert interpretations of the same cases by the Roentgen method in the hands of Dr. Arial W. George — our Hospital Roentgenologist — that I have become much shaken in the confidence which I formerly had that I was making a correct diagnosis by means of my physical examination. Cases of pericardial effusion, which would have been tapped but for the negative findings of the Roentgenograph, not infrequently arise, and these cases cannot all be accounted for by my individual incapacity and lack of skill in physical diagnosis, as in each case I have had as controls the opinions of other examiners whose knowledge and skill are acknowledged to be trustworthy. Of very great and unquestioned value, therefore, is the aid given now by technically good plates and expert interpretation, and of vast importance in the future, when this method of differential diagnosis is perfected, will be the routine use of the Roentgen ray in the hands of expert Roentgenologists.

CASE V. PLATE V. (A. W. GEORGE, M.D.)

Case of pneumothorax in boy of 9 years, taken a year after an attack of hydropneumothorax, showing the cardiac shadow displaced to right, and held there probably by adhesions; shadow of thickened pleura in lower left pleura. An area of either empyematous lung or cavity formation seen in central part of left lung.

CASE VI. PLATE VI. (A. W. GEORGE, M.D.)

Case of osteomyelitis, boy of 8½ years. Process started in epiphyseal line of tibia. Operation delayed and process extended into epiphysis with destruction of bone and sequestrum formation. Capsule of joint thickened as seen by shadow.

The third object of importance to which I have alluded is a branch of special work which I have been engaged in during the past year at the Children's Hospital. This work is to give the normal living anatomy of infancy and childhood, showing the development of the organs and bones at successive periods, with short intervals as of months in infancy and with the intervals in childhood of a length which will be practically useful. Besides showing the changes taking place in all the organs during their growth by months and years they show us the changes so intimately connected with growth in height that it is only a wonder that a complete series of these changes has not been tabulated long ago.

The bones in this series show at what age and period of development the centres of ossification and the epiphyses begin to develop, and the gradual increase until fully developed. The idea of the series is that during the active period of development from infant to child, and child to adult, there are so many varieties under normal conditions that it becomes exceedingly important to have pictures which shall show the different stages. For instance, it is not only necessary for intelligent diagnosis and treatment to recognize diseased conditions, but to be able to compare the diseased conditions of a certain age with those of normal development which would be present at that age. Such a series — as, for instance, in rhachitis — enables the physician to recognize what conditions would be expected at certain stages of the disease by eliminating normal conditions; also the surgeon as to how far the normal conditions, according to the age, should aid in indicating when to operate and to what extent, by keeping in view such conditions. In order to show more clearly the ever changing epiphyses and centres of ossification, I have had some of my plates reproduced for lantern pictures, but it must be understood that these pictures only represent an exceeding small number of the

changes which I have found necessary to tabulate, in order that they can be of practical use to the practitioner, who at any time in a special case may need a particular stage of development to compare with the development of the age he is treating, for the purpose of exact diagnosis, and who will not be satisfied with too long intervals in the plates. It is also to be noted that the pictures on the plates are far more clear than are these reproductions.

Following out the idea of first having a series of normal anatomy to compare with diseased conditions of the same period of development, I have begun the series with the picture of a premature infant. In these normal pictures we have taken, as representing a normal condition, the average of a number of children who, according to a standard of normal external conditions, such as weight, height, development of the teeth, fontanelles and general health, could be taken as representing the normal condition at that age. In order to do this we need at least four or five pictures of the same normal external development to decide upon which picture would be most practical for the use of the physician and the surgeon, and this we have accomplished in this series.*

When we have become familiar with the living normal anatomy of the different parts at the different stages of development, we can, by knowing where to turn for reference in a complete series of plates, or by memorizing these plates, use them as letters of our alphabet, and can, with a proper interpretation of these plates, learn to recognize the living pathological anatomy of such cases.

The series of pictures which I have just shown are simply examples of what the series will represent when completed, for in the short time at my command it would be impossible to describe the many phases and daily changes which, in

* When this paper was read I exhibited 60 lantern slides which, for want of space only, do not appear in this printed paper.

CASE VI. PLATE VII. (A. W. GEORGE, M.D.)

After operation with bone plug in place. Plate demonstrates why operation was a failure. Sequestrum remaining behind plug.

CASE VII. PLATE VIII. (A. W. GEORGE, M.D.)

Case of Villous arthritis in girl of 13 years; capsule thickened and joint full of hypertrophic villi.

even a few cases, are needed to intelligently understand and treat a special case.

The Roentgen method of examination assists and does not replace other methods of examination. Over and over again, however, it corrects and controls an otherwise faulty diagnosis and makes possible a study of the living anatomy and pathology of the different periods of life. In Pediatrics I have found it of inestimable value, for the wonderful recuperative power which exists in children allows great opportunities for a varying treatment, if we can only follow the living, ever changing processes of the disease every few days. Used in this way for diagnosis, the Roentgen ray shows us the primary lesions of the disease, and for treatment therefore, the knowledge obtained by this method is far more important than a knowledge of the terminal lesions found at the post mortem, especially as these latter lesions are often misleading as merely representing terminal destructive processes corresponding to the later stages of a number of different diseases.

Remarks of Dr. Arial W. George, of Boston.

There are certain points which should be noted in regard to the Roentgenology of children. In the first place the technique offers many more difficulties than that which is required in adults. What Dr. Rotch has referred to is especially important, that is, having the first plate taken successfully. Even though a child has submitted quietly when the first plate has been taken, it has become so restless from being restrained that it has been almost impossible to get a second plate, even if it comes on another day.

There are certain conditions which can only be diagnosticated by the Roentgen method. For instance, in normal conditions the epiphyses and centres of ossification are an unknown quantity until an examination has been made by the Roentgen method. In like manner small effusions in the pericardium, and very often the existence of thickened pleura, can be diagnosticated. In other conditions, although the Roentgen method is not the only one which will throw light on the disease, certainly a correct diagnosis cannot be made without it. The truth of this assertion is shown in the differential diagnosis of obscure cases of tuberculosis, osteomyelitis and syphilis.

What I have just said has not included those cases of foreign bodies, which can only be absolutely located by means of the Roentgen method. Also those cases of anomalous conditions of the organs and of the bones. Instance, the absence or imperfect development of the tarsal bones, the absence of ribs, or parts of ribs, and the numerical variations which at times arise in the spinal column.

A subject of especial interest has come to my notice through a fairly large experience in the Out-patient Department of the Children's Hospital: — the injuries to the elbow joint in children. Very little is known at present about these injuries, and what knowledge we have of them

CASE IX. PLATE IX. (A. W. GEORGE, M.D.)

Case of acute infectious osteomyelitis in child of 2½ years, twelve hours after onset. Roentenograph shows a haziness of the outline of the left femur with thickening about thigh. Process went on to general destruction of bone of the entire shaft of the femur. This (Plate IX) illustrates how early changes in tissues and bone can be demonstrated by the Roentgen Ray; as early, in fact, as there are changes in tissue or bone.

CASE IX. PLATE X. (A. W. GEORGE, M.D.)

Case IX. five weeks later. 1.—Areas of suppuration. 2.—Newly-formed periosteal bone. 3.—Necrotic cortical bone. Arrows indicate point of attachment of capsule and periosteum to shaft of femur. Notice that joints have not as yet become diseased, and that infection of the bone is confined within the limits of the periosteum.

is exceedingly imperfect. Yet the results of this lack of knowledge and the unfortunate results of improper treatment are manifest where such a series of injured elbow joints are followed up from beginning to end. The reputation of the surgeon who has charge of these cases will in a vast majority of instances be injured unless he avails himself of such conditions in the elbow as can be seen in the Roentgenograms.

The epiphyses are the most important centres of the skeleton, for on them depend its future development, especially in infancy and childhood, and when these centres are impaired there results far reaching influences in later life. A thorough knowledge of the different changes which take place in even this small area is very important, for these centres are where disease may begin, from which disease may disseminate, where it may lead to disastrous consequences and may leave its permanent marks.

Very little is known of the etiology of the diseases of nutrition and the especially important rhachitic conditions which play such a great rôle in early life, and we also have a comparatively slight knowledge of its early stages. A more exact knowledge, obtained by the Roentgen ray, of the normal development of these parts may lead to the recognition of more definite etiological facts regarding this disease and all other diseases connected with nutrition.

I have never yet found that the radiographs, however misleading they may be through imperfect interpretation, failed to represent a picture of what really exists. The Roentgen method will not demonstrate everything which exists in the body, but it certainly will not show that which does not exist.

In closing my remarks I should like to call your attention to how little regard is paid to the teaching of the Roentgen method of diagnosis in the medical schools. I am sure that great benefit would result from giving such teaching, for it would enable the student to understand the physical conditions existing in disease far better than he does now from simple physical examination of disease.

ARTICLE XXVIII.

SUBACUTE POLYMYOSITIS,

WITH A REPORT OF THREE CASES.

By BENJAMIN T. BURLEY, M.D.
OF WORCESTER.

READ JUNE 11, 1907.

SUBACUTE POLYMYOSITIS.

WITH A REPORT OF THREE CASES.

It was not until the year 1887 that the disease Polymyositis was accurately described. In that year Unverricht (1), Hepp (2) and E. Wagner (3) described cases which have since served as examples of the severe type of this disease. Many physicians have been slow to accept the fact that inflammation of muscles may occur diffusely over the body and constitute a distinct disease, it being long held that muscle became inflamed only from local causes. The recent accumulation of cases, though not great in number, in which general swelling and tenderness of the muscles was the prominent symptom, leaves little room for doubt that they form types of a particular constitutional disease.

For some years the severe form of the disease alone was recognized, but more recently attention has been called to milder types which heretofore have probably escaped notice by being designated as rheumatism, synovitis, etc. While this is not strictly a neurological disease, a considerable proportion of the cases have been brought to light by neurologists.

Symptomatology. — The onset of the disease is usually insidious and gradual, though rarely a case has been reported where the onset was acute with high temperature. A general feeling of malaise and fatigue composes the typical early symptoms. Occasionally there are mild gastric symptoms with anorexia, nausea and some headache. Within a few days the symptoms become more localized

and the patient complains of soreness in the muscles when the extremities are used, occurring rather earlier in the arms. The soreness increases and is often attended by cramps in the muscles. Movement becomes more difficult and produces great fatigue.

Objective symptoms soon manifest themselves. The subcutaneous tissues become swollen, not only over the most affected muscles, but in any part of the body where the skin is not tightly bound down. The œdematous swelling may become very marked, even obliterating the natural contour of a limb. It is usually less towards the extremities of the limbs. Œdema of the eyelids and face has occasionally been marked. This œdema is apparently of inflammatory origin, for it does not pit on pressure and often shows a marked and peculiar redness not unlike that seen in erysipelas. Palpation of the muscles is difficult when this œdema is marked but on its subsidence it is found that the muscles do not feel normal. Their consistency is either hard and distended, tough and inelastic, or more rarely, soft and doughy. Different parts of a muscle may be of variable firmness, comparable to a "hob-nailed liver." After a time the infiltration and consequent shortening of the muscles brings about an approximation of the two ends. In this manner movable parts like the extremities become drawn up at the joints and we get the characteristic attitude of flexed arms and legs in the later stages of the disease. Atrophy naturally follows the destructive process in the muscle and is a prominent feature in chronic cases.

Tenderness of the muscles to pressure is marked in the acute stage, in fact it is difficult to determine whether there is also involvement of the nerve trunks so general is the tenderness. It is considered that the nerves are not involved in a pure case, though polyneuritis and myositis may occur together. The superficial reflexes are all present

unless interfered with by a marked œdema. The deep reflexes vary with the severity and locality of the muscular involvement, the later stages of the disease causing greater interference with the tendon reflexes. The same rule holds as regards electrical excitability, the reaction being normal until the muscle has undergone marked change when gradual diminution and finally abolition of excitability ensue. Fine muscular twitchings are absent. An hyperidrosis of the affected part often occurs.

In general it may be said that the muscles become affected in sequence, the extremities, the muscles of abdomen, neck, chest and finally the throat are involved. The latter paralysis allows the passage of saliva, etc. to the trachea, fostering the development of lung complications which so often form the closing scene in this disease. To the last consciousness is retained, and brain symptoms are absent. The temperature and pulse are both raised, sometimes to high degree, during the acute stages, though mild cases may show little or no increase of temperature. The urine shows no characteristic change though cases with albumenuria have been reported. The blood is usually normal though there have been several cases with accompanying secondary anæmia. The whites are seldom increased. There is *no eosinophilia.*

The course of the disease is very variable, so much so that some authors have made subdivisions into acute, subacute and chronic polymyositis. The subacute course is the most typical. There occur, however, periods of remission in the disease which are apt to deceive the observer. The fatal cases average two or three months in duration, the milder cases may go on for many months before recovery takes place.

ETIOLOGY. — Unfortunately here one can only deal with probabilities. The belief is general that the disease is of infectious origin, but the definite proof is lacking. It must

be said that attempts to discover bacteria in the tissues usually meet with failure, and cultures taken are usually sterile. Bacialli (4) and Georgievskyi (5), however, both report cases where the Staphylococcus Albus was obtained from cultures either of blood or muscle. Bauer (6) also found the Staphylococcus Aureus once. The failure to find bacteria has led to the supposition that some toxic substance was the cause of myositis. Definite information for or against this idea is lacking. Pfeiffer (7) believed the cause was a protozoon, a sporozoon of the group gregarinæ. He failed to find it however in specimens submitted by Strümpell (8) and Unverricht.

The argument in favor of an infectious origin is strengthened by the onset with malaise, the fever and the oft-occurring splenic enlargement. Cases reported by Herz (9) and Lewy (10) suggest not only the infectious but also the contagious nature of the disease. Herz describes two mild cases in girls occupying the same room, one case slightly preceding the other. Lewy made a diagnosis of polymyositis in three members of the same family.

Age and sex apparently have no bearing on etiology. In 1895 Jacoby (11) wrote that two thirds of the published cases had been males. All ages had been affected except childhood. Since then five cases have been reported in children, two following some infectious process.

Pathology. — The post-mortem findings in these cases are variable, depending largely upon the stage of the process. A majority of the cases, however, have reached the later or destructive stage, the early or inflammatory stage being less in evidence except when a specimen of muscle is removed during life. The signs of inflammation including swelling, infiltration, transudation and hemorrhage are found early affecting largely the connective tissue, in part the parenchyma. It is characteristic of the disease that certain bundles and certain fibres of a bundle are unaffected

by the process. With the progress of the disease attempts at regeneration are commonly seen in the great increase of nuclei including mitotic forms, while, at the same time, the anatomical picture of destruction of fibres is prominently seen. This appears as granular and, later, waxy degeneration of the fibres, vacuolation, a great increase of cells and fat globules, greatly thickened perymysium becoming myxomatous or fibrous in structure, in fact a tendency to the embodiment of the former specific tissue into a connective-tissue structure.

The nerves have been shown to be involved in a number of cases, notably those of Senator (12), but from their distribution and from the considerable number of normal fibres found in the sections it seems probable that the process is not primary in the nerves. The amount of hemorrhage between the muscle bundles is variable. It may be very great, predominating in the sections and giving rise to the name polymyositis hemorrhagica. (Lorenz) (13).

Diagnosis. — Once one's attention has been drawn to this disease, he will seldom fail to make the diagnosis. The differentiation from trichinosis and from polyneuritis presents the chief difficulties. Other affections that at times resemble this condition are phlebitis, periostitis and osteomyelitis, rheumatism and pyemic abscesses.

Leube (15) and other diagnosticians say that it is impossible to distinguish polymyositis from trichinosis by the symptoms alone, an examination of specimens of muscle being necessary. Since 1897, however, when Brown (14) called attention to the constituents of the blood in trichinosis, especially the eosinophilia, we have had a most valuable yet unheralded aid to diagnosis. The accumulated evidence goes to show that cases of trichinosis have constantly an increase of eosinophiles from 10 to 50 per cent., with a corresponding decrease of the neutrophiles. I have seen the report of no such blood count in polymyositis, in fact

the differential count is usually normal. Leucocytosis too is more common in trichinosis. A history of eating uncooked pork of course points to trichinosis. Primary gastric and intestinal symptoms and involvement of the muscles of the eye and diaphragm are more characteristic of trichinosis.

As against a polyneuritis the facts which point toward a polymyositis are: Tenderness over the affected muscles rather than along particular nerves. Inflammatory œdema involving both the muscles and the cutaneous tissues. Absence of anæsthesia or paræsthesia. The muscular swelling followed after a considerable time by atrophy, often with contracture.

It happens that the mechanical interference with the reflexes and electrical reactions found in polymyositis may at times closely resemble the changes found in polyneuritis, thus rendering them of uncertain value in differential diagnosis. It must be remembered also that the two affections may be associated.

Syphilitic myositis is another rare affection that may be mistaken for polymyositis. It occurs in three forms — the gummous, the diffuse and the combined (Herrick) (16). It is accompanied by evidences of syphilitic infection, usually affects only a small number of muscles, and has not the splenic tumor or the exanthem of polymyositis.

Prognosis. — The prognosis, formerly almost always regarded as fatal, now has to be modified with the recognition of the milder cases. The more general the involvement of the muscles and cutaneous tissues, with a persistent temperature, the graver the prognosis. The most dangerous single symptom is involvement of the muscles of deglutition, whereby the saliva is not diverted from the respiratory tract and lung complications occur. Myocardial involvement, though rare, adds greatly to the danger. Long drawn out cases tending to recovery are apt to have some muscles permanently atrophied.

TREATMENT. — Attempts to cut short the course of the disease by any specific form of treatment have heretofore been of little avail. Antipyretics and salicylates have somewhat relieved the pain and soreness of the early stages. Hot baths and fomentations had been moderately used before, but in 1903 Oppenheim (17) came forth with glowing accounts of five cases which led him to confidently advocate the systematic use of diaphoresis. His method is to apply hot air, then wrap in blankets, giving hot drinks and aspirin. The patient is thus sweated for an hour at least every second day. Thermo-massage is used on the alternate day. Later simple massage and electricity are used.

Oppenheim considered this treatment a great factor in the recovery of his cases, though it did not serve me as well in the one case on which I had an opportunity to try it. The test of time will alone determine its value. My cases will now be presented in the order of their occurrence.

CASE I: On June 11th, 1904, Dr. G. S. Clark asked me to see a boy 15 years old, who presented the following history: He was a member of a healthy English family which included nine other children. The family were characteristically small of stature; this boy, who had grown rapidly the last two years, being rather the largest of the family. The patient had been well up to six months ago when it was said that he fell on the ice, striking the lower end of his coccyx. No particular complaint was made at the time and no paralysis ensued, but within a few days it was remarked that the boy was less energetic and more easily tired. Sometime later it became difficult to make the boy do such chores as bringing up wood or carrying water, he complaining of soreness in the muscles of the back and arms. Six weeks ago the eyelids began to swell, then the face, especially the upper lip. Both arms and hands then became œdematous, stiff and tender to pressure, though there was no real pain. This œdema gradually extended to back, abdomen and legs, until at the time of my visit there was practically complete anasarca. Muscular

weakness has been progressive, so that now the boy can sit up but little, and can scarcely stand alone. There has been no sphincter disturbance at any time. The knee-jerks are said to have been absent for one month. At no time has there been any gastric disturbance. The appetite has been good and the bowels regular. Slight trouble with swallowing solid food developed a week ago; since then the diet has been liquid and soft solid. The temperature when taken has been found to be from 99 to 100.

Physical examination showed the boy to be well developed and apparently well nourished. Anasarca was complete, except for the plantar surfaces of the feet. The œdema was especially marked over the face and buttock, the projection of the eyelids being fully one half inch, and cutting out all light from the eyes. No discoloration or rash was present on the skin. Mucous membranes have fairly good color. On palpation the skin did not pit, as was expected, on pressure, the muscles were moderately tender and felt boggy. The large flexor muscles especially felt tense and of irregular hardness. The muscles along the upper part of the spine were very tense. There was little or no impairment of tactile sensation. Pain and muscle senses were also normal. There was some hyperæsthesia, especially along the spine. The right pupil was very slightly larger than the left, but both reacted normally to light and distance. The patella reflexes were lost, as were also all the superficial and deep reflexes except the plantar, which was normal. Babinski absent. The heart was not enlarged and the sounds were normal, except for a slight bruit in the neck. On exertion the heart became very rapid, causing some dyspnœa. The urine was normal. The boy was mentally clear. Blood examination showed 60 per cent. hæmoglobin. No increase of eosinophiles of white cells and no abnormal forms of red cells.

The tentative diagnosis of polyneuromyositis was made and a grave prognosis given.

The case was next seen on July 26th. During the interval the patient had been on a liberal semi-solid diet and a preparation of iron, arsenic and strychnia. In good weather too he had been carried out doors, and had spent much of the day under a tent. One month ago the œdema

began to dis ppear, beginning at the upper part of the body, until four days ago it was absent. There had been marked disturbance with the muscles of deglutition until three weeks ago, but since then considerable improvement in that respect. The arms and legs have become more flexed, so that walking is impossible. The appetite is still good. Examination of the muscles shows that they have lost their boggy feel, and are much thinner than before. The biceps and hamstring muscles, especially, are shortened and feel hard and fibrous. The reflexes are the same as at the last visit. There is now no skin hyperæsthesia. Today there is considerable tenderness of the flexor muscles of the arms and legs, and slight œdema especially about the ankles. This may possibly be due to the fact that the attending physician has allowed more vigorous massage for the last three days.

At my final visit, Sept. 10th, I found that during August there had been apparent improvement in the use of the arms, but in all other respects the boy had gradually grown weaker. There has been steady and gradual wasting of the muscles and flesh over his whole body. In fact I never saw a more striking contrast than the pathologically plump figure of three months before and the shrunken skin-covered skeleton of today. The legs and arms were drawn to a right angle and the affected muscles felt only like shrivelled cords. The patient is now scarcely able to move any muscles except of the neck and face. The scalp and skin is dry and scaly, and there is marked alopecia especially at the sides of the head. Saliva collects and runs out of the mouth, for it cannot be swallowed. There has been increasing difficulty in nourishing the boy, and now for three days it has been impossible to move the bowels. The heart is rapid with no murmurs. The lungs are clear. Nothing abnormal found on palpation of the abdomen.

After this visit the patient is said to have failed steadily, retaining his consciousness, until he died of respiratory failure two days later, Sept. 12th, 1904. It is to be deplored that the attending physician was unable to get permission for an autopsy.

CASE II: Mrs. O. is now 30 years old and has three healthy children. Her parents both died in late middle life, the father of "brain fever," the mother of pneumonia. She has three sisters who are well, and one who has suffered for a year with inflammatory rheumatism. The patient has generally been well, except for attacks of diphtheria at 20 years and bronchitis at 25 years. From both of these she made a good recovery.

On April 9th, 1904, just as her husband was recovering from an attack of the grip which had kept him in the house two weeks, the patient first noticed aching pains becoming general over the body. There was no disturbance of the stomach, and Mrs. O. asserts that she had eaten no pork for a long time. Headache and any marked feverishness were absent. The patient felt tired all of the time, but kept about. At the end of a week some swelling occurred about the eyelids and upper part of cheek, and rapidly extended over shoulders, back, arms and legs. The swelling was brawny and did not pit on pressure. The feet did not swell, but the hands were swollen even to the finger tips, where there was some formation of pus about the nails. Within a few days, the soreness of the muscles persisting, the patient found she was unable to lift up or hold her baby, and if she stooped bending her knees she was in danger of falling. Within two or three weeks the swelling had largely disappeared, except about the flexor muscles of the arms and legs. At about this time the muscles of deglutition became affected so that only liquids could be swallowed without difficulty. The impairment of function in these muscles did not lead to further serious symptoms and their power gradually returned in a few weeks. Dr. S. B. Woodward, who had charge of the case in June, always found some elevation of temperature, the highest being 102 degrees, and advised her removal to a hospital. From June 24th to August 16th, Mrs. O. was a patient at Memorial Hospital on the service of Dr. R. W. Greene. At Dr. Greene's request I saw the patient early in July. She was of medium height and well nourished, did not appear to be very ill and was not confined to her bed. Color was good. Skin dry and rather scaly. Scalp poorly nourished and hair falling out. Arms were held partly flexed, and

CASE II.

LEFT BICEPS MUSCLE.

Cross section. Mag.=150. Early stage of interstitial polymyositis showing invasion of the muscle bundles with many mononuclear cells from the direction of the fascia. Lower part of section shows normal muscle.

could not be fully extended. Attempts to do so produced pain and brought into relief the biceps muscles which were of irregular, abnormal hardness. The surface temperature over these muscles was evidently increased. The cutaneous tissues were somewhat swollen. The supinators, especially the left, were similarly affected. The flexors of the legs were also hard, thickened and apparently shortened so that complete extension was painful. There was some swelling and tenderness, too, above the knees in front. Sensation was everywhere unimpaired. The pupils were equal and reacted normally to light and distance. The elbow jerk was present but slight. Knee jerk not obtained on either side. Achilles reaction normal. No ankle clonus. No Babinski. Superficial reflexes were present, but not increased. An electrical examination was not made at this time. The affected muscles did not react to mechanical stimuli. There was no paralysis, but lessened power in the flexor muscles of the arms and the flexors and extensors of the legs. The urine at this time was normal in color, sp. gr. 1020. No albumen or sugar. Blood examination showed: Hgb. 80%. Reds, 4365000. Whites, 8000. Differential count normal, no eosinophilia being present.

During the patient's stay in the hospital she was under no specific treatment. She was given general tonics and occasionally the static breeze. She took moderate exercise in the open air. About a week after entrance the quantity of urine was somewhat less and an occasional hyaline cast appeared in the urine. Probably on this account she was kept on a vegetable diet during the rest of her stay. In August, having made no improvement, she was taken by the interne to consult a leading physician in Boston. This physician wrote his opinion that the case resembled trichinosis, but the course of the disease and the normal blood examination were against this diagnosis. He inclined to regard it as a peculiar type of polyneuritis. He recommended examining a piece of muscle and advised general tonic treatment. Two weeks later the patient was discharged from the hospital unimproved.

Sept. 2d, Mrs. O. came to the Neurological Dept. of the City Hospital and consented to the removal of a piece of muscle. Under primary ether the House Surgeon removed a piece of the left biceps muscle 2 cm. long by 1

cm. in thickness. In cutting, the muscle felt decidedly tough and it bled more than normal. One cat-gut stitch closed the muscle edges, and three of silk-worm gut closed the skin. Part of the specimen was put in Zenker's fluid and part in alcohol. The gross specimens appeared mottled; grey and red. For the subsequent pathological report I am indebted to Dr. Hunt:

"In the main the sections show little departure from normal striated muscle. In several, however, there appear a few areas of hemorrhage and of round-cell infiltration into perimysium extending along endomysium into peripheries of the neighboring muscle fasiculi, where the muscle fibres show varying degrees of disintegration and hyalin change. Many fibres show a separation of fibrillæ, take the stain poorly and are penetrated by round cells; while others are dense and homogeneous, show no striation, and stain deeply with eosin.

Sections stained by Loffler and Gram-Weigert methods show no bacteria or parasites.

Diagnosis:—Non-suppurative, hemorrhagic interstitial myositis."

The patient was given Quinine bi-sulphate gr. v. t. i. d. and put on a full nitrogenous diet. The examination at this time showed no marked change from two months before, though the knee-kerks were more easily obtainable. There was very marked alopecia. The wound of the arm healed by first intention and the stitches were removed. I did not see the case again until Oct. 27th. She had been keeping on with the treatment but on account of illness in the family had been unable to follow directions about resting. She was taking care of her house and three children. In spite of this she seemed to have improved. The skin was much less dry. The hair was growing in thickly. The arms could be extended almost fully without pain. The supinators had apparently lost their induration. Flexors of legs and biceps were still involved and tender to deep pressure. All the muscles react readily to the faradic current but there is no abnormal irritability. The patient's appetite has improved and she has menstruated for the first time during this illness except for a slight trace in July.

Gradually the stiffness and weakness of the arms and legs disappeared and she was able to attend to her house-

A

B

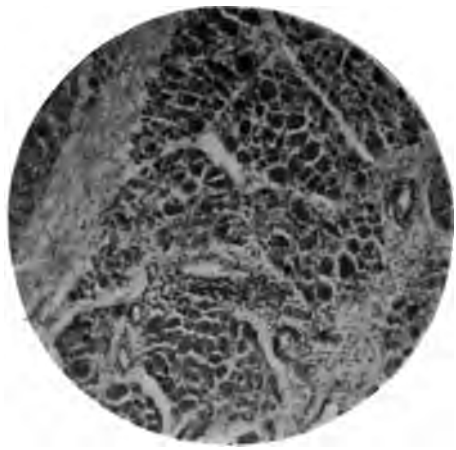
CASE III.

RIGHT BICEPS MUSCLE.

A.—Cross section. Mag.=40. Shows separation of bundles by œdema and excess of connective tissue. Pallor and shrinking of fibres at borders of bundle especially.

B.—Cross section. Mag.=400. Shows varying size of muscle fibres with marked granular degeneration and vacuolation. Excess of nuclei with occasional mitotic form.

C



D

CASE III.

C.—Cross section. Mag.=80. Shows marked infiltration of the perimysium and endomysium with round cells. Disintegration of muscle fibres going on; comparatively few fibres staining deeply.

D.—Longit. section. Mag.=80. Shows narrowing and crinkling of fibres, areas of vacuolation and waxy degeneration, also excess of connective tissue, with round-cell infiltration.

hold duties. She passed through a normal pregnancy and had a healthy child born June 28, 1905. There has been no recurrence of her trouble.

CASE III: A Swede 31 years old and married is said to have been the strongest man in the wire mill, where he was a foreman. He was referred to me by Dr. W. H. Rose and gave this history. There was no trace of chronic illness in the family and, except for an attack of malaria a year previous, the patient had remained well up to August, 1904.

At about this time, without any history of digestive trouble, he began to complain of lameness and soreness in the anterior aspect of both thighs. A little later a similar condition arose in the calf muscles and, later still, the back became affected. There was some general malaise and the patient felt tired all over so that he left his work. After a three weeks rest he felt somewhat improved and went back to work. During his stay at home a considerable puffiness of the face with redness was first remarked. The lameness now extended to the arms and neck, muscular exercise became painful and the muscles were rather tender to pressure. The patient has now been at home six weeks and has gradually failed in strength. There has been profuse hyperidrosis with little fever.

Physical examination Dec. 17, 1904.—The patient appeared to be a remarkably well developed and muscular man, yet he had considerable difficulty in getting up the steps to my office. Beads of perspiration stood upon the face and neck. The face was slightly erythematous and puffed. There was a brawny œdema over the neck and arms, also slightly over the thighs. Some tenderness is elicited over the affected parts but this tenderness does not follow nerve trunks. No loss of sense of touch, pain, temperature or position. Full extension of arms or legs is difficult and causes pain in the flexor group of muscles. The hand grasp is weak, muscle fatigue coming on rapidly.

The pupils are equal and react alike to light and distance. The triceps, quadriceps, achilles and plantar reflexes are present. Babinski absent. The muscles did not respond abnormally to mechanical stimuli. Irritability to both galvanic and faradic currents was normal except at œde-

matous areas, where it was slightly diminished. The abdomen was full and muscular, making examination difficult, but the spleen seemed to be just palpable.

Heart apex in fifth interspace on nipple line. No murmurs. Pulse 90 and of fair volume. Temperature 99°F. The urine was negative; color, high. Reaction, acid. Sp. gr. 1024. No albumen or sugar.

Blood Examination: Red cells, 4300000. Hæmoglobin, 80%. White cells, 9000.

Polymorphonuclear leucocytes,	70%.
Lymphocytes,	25%.
Basiphiles,	4%.
Eosinophiles,	1%.

Aided by my experience with two other cases, the diagnosis of Polymyositis was readily made and the patient's removal to the Worcester City Hospital advised. The treatment adopted at the hospital was, briefly, rest in bed with a short period for exercise, a diet composed largely of nitrogenous food with a large amount of liquids, diaphoresis, thermotherapy and quinine sulphate gr. v after each meal.

During the next month there seemed to be slight improvement, though on some days the œdema about the face and neck would be well marked. The biceps and quadriceps muscles felt brawny and inelastic. The patient's appetite remained good and he got normal sleep. Control of the bladder and bowels was normal.

Towards the latter part of January, 1905, the swelling of the right arm increased considerably and the hand and arm were extremely painful on motion. Hyperæsthesia along nerve trunks and functional weakness indicated that the brachial nerves had become involved.

The patient had had one or two attacks of epistaxis earlier, but on the 22d of January the flow was so free that the nares had to be sprayed with adrenalin and packed. There had developed a slight systolic heart murmur near the apex but not transmitted outward.

February 9.—Treatment by drugs, diaphoresis, etc., does not seem to stay the progress of the disease. The patient has marked swelling about the throat and begins to find it difficult to swallow food. Examination of the throat by Dr. Estabrook shows marked infiltration and weakness

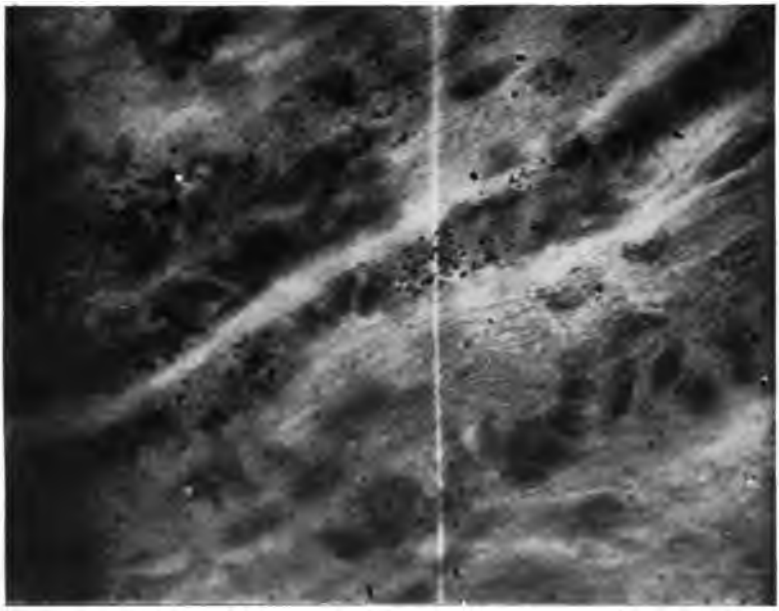
E

F

CASE III.

E.—Longit. section. Mag.=200. Shows longitudinal cleavage. Proliferation of nuclei, resembling giant cells. Loss of striation in all muscle fibres.

F.—Cross section. Mag.=40. Shows wide separation of muscle bundles by œdema. Apparent disintegration of peripheral fibres earliest by pressure or toxines. Fibres in centre more nearly normal.



CASE III.

Longit. section. Mag.=1200. Shows clumps of deep staining bodies (cut by white line) resembling bacteria, but probably the result of granular degeneration of the sarcoplasm.

of the muscles of the pharynx and larynx. Preparation for tracheotomy, if necessary, is made.

February 12.—After progressive difficulty in respiration and swallowing of saliva, the patient suddenly had a choking fit in the night and died before the physician reached his bed. He was practically clear mentally until the end.

Autopsy was not allowed, except the removal of a portion of the right biceps muscle. In removal the subcutaneous tissues were found infiltrated with serum and the muscle was pale, yellowish and friable in places. The excised muscle, when put into alcohol, appeared mottled gray and red on the cut surface.

Some of the results of the pathological examination of specimens from this muscle are shown in the accompanying photographs. The specimens were hardened either in Zenker's fluid or alcohol, imbedded in paraffin or celloidin and stained with hæmatoxylin and eosin, methylene blue and eosin or by the method of Van Gieson. Sections stained by the method of Gram-Weigert and carefully studied failed to give positive evidence of bacteria in the tissues.

Following the classification of Lorenz, the above cases would fall under the head of "Dermatomyositis," yet they are so characteristic in their insidious onset and slow course and general involvement of the muscular system that I have chosen the title "Subacute Polymyositis." Cases I and III follow out the classical symptoms of the most severe form of dermatomyositis, except that there was little erythema, and both died with the usual respiratory involvement. Circumstances beyond my control prevented an autopsy in Case I, which is to be the more regretted because in this case the disease ran its full course from the insidious beginning through the fulminating stage with most marked inflammatory œdema to the stage of extreme wasting and atrophy. If the theory is correct, which seems to be borne out by pathological reports, that the disintegration of muscle taking place as a result of invasion by toxines or other products, is followed, with the subsi-

dence of inflammation, by a low grade of connective tissue formation, then, in Case I, I feel confident that practically no normal muscle structure would have been found but scarcely more than strips of connective tissue. At any rate my last examination by palpation of this case would warrant that conclusion.

We find Case III, however, dying of asphyxiation with the muscles about in the middle stage of the slow process. Some bundles show the earlier stage with great infiltration with leucocytes and strange nuclei, some are marked by the excess of serum and areas of hemorrhage, some by granular and hyaline degeneration with vacuolation, but few show any advanced degree of organization of connective tissue.

Case II shows chiefly the beginning invasion of the bundles by leucocytes and round cells.

The etiology of these cases is unknown. Cultures taken from Cases II and III gave no growth on various media, and no definite bacteria or protozoa could be stained in the tissues. It is a remarkable coincidence that all these cases appeared in the same city in the same year, but each case was three miles from any other and in no way associated.

Possibly, in view of the fact that none of these cases were diagnosed for several weeks until I saw them, and that Case III was referred to me only after my report of the others at a medical meeting, the malady may be more common than is supposed. The very small number of typical cases reported, however, bespeaks their rarity. Lorenz could collect from all sources only 15 cases which he classified as Dermatomyositis. In 1904 Steiner (18) had collected 28 fairly typical cases, giving a resumé of the pathological examinations, when made. Since that time several authors have reported cases of some form of polymyositis, but I have been unable to satisfy myself that more than one of these belongs to the type of severe dermatomyositis,

CASE III.

This photograph was intended to show the œdema about the eyes, face and neck, also the brawny swelling of the arms which prevents their extension; but, on account of the perspective, it fails adequately to depict the actual condition.

such as two of my cases present. This case, the first to be reported from Russia, is by Preobrazhenski and Margulis (19).

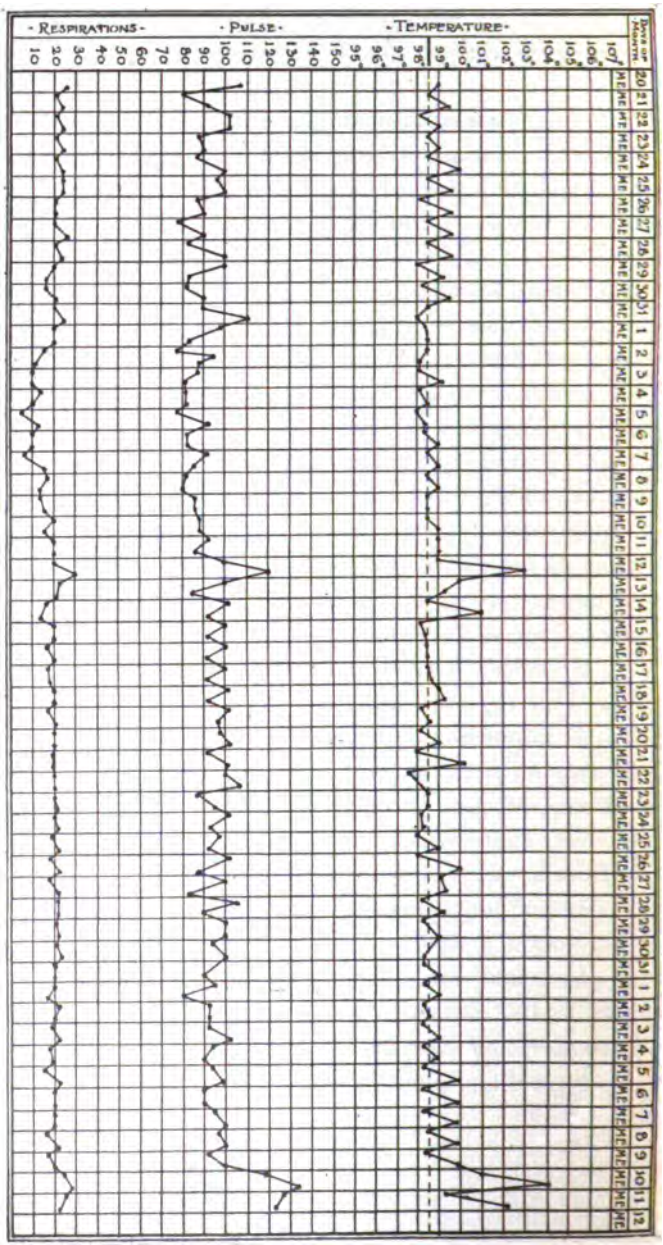
The young woman affected has had the disease coming on insidiously for six months when examined. There is some œdema and general involvement of the muscles, especially of the arms and legs. Epistaxis is a prominent symptom. A section of muscle is excised from the leg which shows the changes of polymyositis. In spite of treatment the disease progresses and the woman dies of laryngeal palsy three months later. There is a complete autopsy report of various muscles and the changes are similar to those described in Case III.

Cases reported by J. Baer (20), Bevill (21), Méry, Terrien and Génevrier (22), Murrell (23) and Sick (24) are in some way atypical. Sick reports three fairly severe and six mild cases with symptoms of polymyositis occurring in epidemic form, but the cases are too acute and short-lived to belong to this group.

Some Japanese authors have written of forms of polymyositis occurring in Japan, but, from the literature obtainable, I do not find the cases of the subacute type. Ito and Konaka's (25) ten cases of myositis apparently arose from direct infection through the skin or tonsils and caused local suppuration, necessitating operative procedure. Hnátek (26), among others, has described a case more typical of polymyositis hemorrhagica.

In conclusion, I wish to express my indebtedness to the several physicians for referring to me these cases, and especially to Dr. Ernest L. Hunt, Assistant Pathologist of the Worcester City Hospital, to whom all credit is due for the pathological and photographic work in Case III.

CHART OF CASE III.



BIBLIOGRAPHY.

1. Unverricht.—Ueber eine eigenthümliche Form von acuten Muskelentzündung mit einem der Trichinose ahnelnden des Krankheitsbilde. München. med. Woch., 1887, xxxiv, 488.

2. Hepp.—Ueber Pseudo-trichinose, eine besondere Form von acuter parenchymatöser Polymyositis. Berl. klin. Woch., 1887, xxiv, 297 and 322.

3. Wagner, E.—Ein Fall von acuter Polymyositis. Deutsches Archiv. f. klin. Med., Leipzig, 1886-7, xl, 241.

4. Bacialli.—Contribuzione allo studio della patogenesi e delle alterazione istologiche del tessuto musculare nella polimiosite acuta primaria. Il. Policlinico, 1902, ix, 16.

5. Georgieoski.—(A Case of Acute Homorrhagic Polymyositis.) Bolnitsch. Gaz. Botkina, 1900, xii, 696. Also, ref. in Boston Med. and Surg. Jour., 1902, cxlvii, 313.

6. Bauer.—Ein Fall von acuter hämorrhagischer Polymyositis Deutsches Archiv f. klin. Med., 1899, lxvi, 95.

7. Pfeiffer, L.—Die Unverricht'sche Polymyositis gregarinosa oder Dermatomycosis acuta. Die Protozoën als Krankheitserreger, Jena, 1891, 2 Aufl., 208.

8. Strümpell.—Zur Kenntniss der primären acuten Polymyositis. Deutsche Ztschr. f. Nervenh., 1891, i, 479.

9. Herz.—Ueber gutartige Fälle von Dermatomyositis. Deutsche med. Woch., 1894, xx, 790.

10. Lewy.—Zur Lehre von der primären acuten Polymyositis. Berl. klin. Woch., 1893, xxx, 475.

11. Jacoby.—Subacute progressive Polymyositis. Jour. Nerv. and Ment. Dis., N. Y., 1888, xv, 697.

12. Senator.—Ueber acute Polymyositis and Neuromyositis. Deutsche med. Woch., 1893, xix, 933.

13. Lorenz.—Die Muskelerkrankungen. Wien, 1898, 120.

14. Brown.—Studies on Trichinosis. Johns Hopkins Hosp. Bull., Baltimore, 1897, viii, 79.

15. Leube.—Medical Diagnosis. Trans. of sixth German Edition, 1804, 769.

16. Herrick.—Polymyositis acuta, with report of a case presumably of syphilitic origin. Am. Jour. Med. Sc., 1896, cxi, 414.

17. Oppenheim.—Ueber die Polymyositis. Berlin klin Woch., 1903, xl, 381 and 416.

18. Steiner.—Dermatomyositis, with report of a case, etc. Jour. of Exp. Med., Baltimore, vi, 407.

19. Preobrazhenski and Margulis.—K uchenigu o poliomiozitie., J. nevropat. i psikhiat., Kossakova, Mosk., 1904, iv, 841. Also, trans. Rev. de Méd., Paris, 1904, xxiv, 765 and 851.

20. Baer, J.—Ueber akute Polymyositis. Münch. med. Woch., 1904, li, 151.

21. Bevill.—A case of polymyositis with a rare complication, blindness. St. Louis Med. Rev., 1904, l, 290.

22. Méry, Terrien and Génevrier.—Polymyosite infectieuse aiguë bénigne chez un enfant de neuf ans. Bull. et mém. Soc. méd d. hôp. de Paris, 1904, 3s., xxi, 1115.

23. Murrell.—Dermato-myositis or Acute Polymyositis. Med. Press and Circ., London, 1905, n.s., lxxix, 207.

24. Sick.—Akute rezidevierende Polymyositis in epidemischen Auftreten. Münch. med. Woch., 1905, lii, 1092 and 1152.

25. Ito and Konaka.—Juntendo Iji Kenkiu Kwai Zasshi., Tokyo, 1903, 883. Also, trans. Deutsche Ztschr. f. Chir., Leipzig, 1903, lxix, 302.

26. Hnátek.—(Case of Polymyositis with Hemorrhages). Trans. Wien med. Presse, 1905, xlvi, 917.

ARTICLE XXIX.

THE OPERATIVE TREATMENT OF VARICOSE VEINS OF THE LOWER EXTREMITY.

By JOHN T. BOTTOMLEY, M.D.
OF BOSTON.

READ JUNE 11, 1907.

THE OPERATIVE TREATMENT OF VARICOSE VEINS OF THE LOWER EXTREMITY.

Not alone to the greater ills and affections that in themselves endanger human life should our professional care and attention be directed; the lesser ailments, rarely fatal but frequently disabling, call for relief and justly claim their due share of our notice. It is with this thought in mind that I bring to your attention in this paper the subject of varicose veins of the leg. I need make no apology, I trust, for my choice of topic. Recognized for centuries, wide-spread in its field, frequently crippling in its effects, attacking most often the wage-earning class, this affection of the venous system is worthy of our best endeavors for its relief. Its treatment usually falls within the province of surgery, and it is through operative procedures alone that permanent and satisfactory results can be obtained in the very large majority of cases.

No lengthy discussion of the subject will be undertaken, but it may not be amiss to recall a few important facts with regard to the anatomy, etiology, symptoms and pathology of the affection before taking up the operative treament.

There are two systems of veins in the lower extremity. In the leg each main artery has two accompanying veins which unite at the lower border of the popliteal space to form the popliteal vein; the latter in its turn becomes the femoral.

The internal and external saphenous veins with their branches form the superficial system. A detailed description is unnecessary. It is important to note that the internal saphenous has free communication with the deep system through the deep fascia. It also communicates freely with the external saphenous in the foot and leg. The external saphenous has but one important branch; it is given off just before the parent vein empties into the popliteal and, after turning around the inner border of the thigh, anastomoses with a large branch of the internal saphenous to form a well-marked connection between the two larger veins. It frequently shows a varicose condition.

The external saphenous has from six to twelve valves, and the internal from eight to twenty. This is a most important arrangement. Under normal conditions the blood pressure in the veins is distributed in such a way that at any given point of the vein wall the pressure is only that caused by the weight of the blood column between it and the next higher valve. With incompetent valves the story is quite different. Above Poupart's ligament there is no valve in the femoral, external iliac or inferior cava veins; hence, there is nothing to prevent the pressure of the blood in these vessels from being communicated to the internal saphenous, and the latter must sustain the weight of the blood column between itself and the heart. This is a relatively serious matter, when the valves of the vein are for any reason incompetent.

The etiology of varicose veins is obscure. Most of the so-called causes are really neither primary nor originating; they are simply exciting or aggravating agents. Many cases, indeed, are seen in which are present none of the conditions ordinarily mentioned as causes. The affection, as seen in our out-patient clinics, is most common in patients in the third and fourth decades of life. It must be remembered, however, that these patients seek aid not at

the first appearance of symptoms, not when the varicose condition is first noticed, but only when it becomes troublesome. Kennedy[1] in his report of twenty-seven cases informs us that in no fewer than six the varicosity appeared at the age of ten or earlier, and that in eleven cases it was noticed between the tenth and the twentieth years. Such facts give credibility to the opinion of Bennett,[2] who calls attention to the frequent early appearance of the condition and maintains that the most probable cause is a congenital defect by reason of which the valves are not normally competent; this incompetency of the valves, says Schambacher,[3] exists because of congenital weakness of the vessel wall, due to the irregular development and arrangement of its elastic and muscular elements. The probability of a congenital etiological factor is further increased by the fact that the condition may exist in both extremities at the same time or in either alone. Of 864 cases, including those of Bennett, Miller, Goerlich and the Carney Hospital series, both extremities were affected in 414, the left alone in 252, and the right alone in 198. Heredity, too, is said to have an etiological influence. There is, moreover, a relatively small group of cases—the "inflammatory" of Miller,[4]—in which a pretty definite relation can be established between the varicose condition and a preceding phlebitis, consequent upon pregnancy, typhoid, erysipelas, pneumonia, etc. Of Miller's 128 cases 40 are classified under this head. Trauma is unquestionably a frequent exciting cause.

Little need be said of the pathology. As we see the cases, the gross pathology is usually evident. The fundamental pathology rests on the facts that, whatever the cause may be, there exists stasis in the veins of the lower extremity, that the valves are incompetent, and that the veins most often affected run in the subcutaneous fat where there is but little resistance offered to their dilatation. With the late pathology, the simple phlebitis, the organ-

ized phlebitis, the periphlebitis, the ulceration, the eczema, etc., we are all familiar.

The symptons vary in severity. Some cases are subjectively symptomless; in many, on the other hand, the symptoms are so severe as to seriously cripple or even incapacitate the patient; the former seldom present themselves for treatment; of the latter, many examples are seen.

What does surgery of the present day offer in the way of radical treatment? How much hope of permanent cure can we promise these cases? Though numerous operative procedures have been proposed for its relief, those in use to-day have as their foundations one of the following methods: (1) The Trendelenburg, (2) the Schede, or (3) the Madelung. These operations, modified or combined, are conceded to be the most effective of all.

1. Trendelenburg[3] in 1890 published his method of division of the internal saphenous between ligatures close to the saphenous opening. This prevents the transmission of the weight of the blood column in the femoral, iliac and inferior cava veins down to the internal saphenous with its incompetent valves. The procedure has been modified and now excision of about two inches of the vein is practised instead of simple ligation. While there can be no doubt that in many, especially the early, cases, his method gave good results, yet recurrences have been reported in percentages varying from 6% (Remedi)[6] to 15% (Faiszt)[6] and even 22% (Perthes)[6]. Miller[4] from a study of reported cases and of the Johns Hopkins Hospital series concludes that the cures average about 75%. The percentage of recurrences increases with the length of time after operation, rising in the Johns Hopkins Hospital series from 11% to 37% from the 4th to the 8th year.

2. In the Schede[7] operation all veins superficial to the fascia are divided between ligatures, through an incision

encircling the leg in its upper third. Von Wenzel,[6] a former pupil of Trendelenburg, modified the procedure by making a similar incision at the junction of the middle and lower thirds of the thigh and dividing the superficial veins at that level. A "horse-shoe" incision has also been used in place of the complete encircling incision. These operations attempt to attain their objects by forcing the blood to get out of the leg and into the femoral vein by way of the deep venous channels lying under the fascia. In a small series of nine cases at Johns Hopkins, 67% had recurred at the end of ten years.

3. Madelung[8], through a long incision from which the skin is turned back a hand's breadth on either side, excises practically the entire internal saphenous vein and a portion of its branches. Many modifications of this method have been offered, the chief ones being subcutaneous extirpation of the vein or excision of small portions throughout its length. 90% of cures is claimed for it.

The Trendelenburg operation is easy of performance and practically free from danger. But often it does not fulfil the conditions necessary for a radical cure. Two factors militate against its success and, in a way, against that of the Schede method. (*a*) First, the internal saphenous vein has free communication with the deep system and also with the external saphenous. This communication may play an important part in the prevention of a cure; for Bennett believes that varicosity, stasis and consequent increased pressure in the deep veins often coexists with a like condition in the superficial. It is evident that frequently in the Trendelenburg operation no barrier is offered to the transmission of this increased pressure and its effects to the superficial veins. In fact, consequent to this operation, enlarged veins either appear or, if present at the time, increase in size. We have noted that, even after extensive extirpating operations, some of the venous branches which

were not removed have later become enlarged; this enlargement could take place only because of back pressure from the deep circulation. (*b*) The second factor in recurrence is that restoration of the superficial venous circulation sometimes takes place after ligation. (Miller,[4] Ledderhose,[9] Goerlich[10].) This may occur in three ways: (1) by anastomosis around the scar, (2) by formation o varices in the scar, or (3) by union of the ligated stumps with formation of an intact vein. This is an interesting phase of the subject, but we can not discuss it in a paper of this length.

Occasionally, though rarely, gangrene of the foot may follow the Schede procedure. Much more common sequelæ are œdema of the leg and either hyperæsthesia or anæsthesia (more frequent) below the scar because of section of the subcutaneous nerves. Von Wenzel[6] claims that his operation is followed by no such ill results. In three cases reported by Georgi,[8] no anæsthesia appeared, despite the fact that in all three operations he saw and preserved just one nerve filament. We have had no experience with the method, but beyond the fact that it shuts off the superficial venous circulation at a higher level than does the Schede, it seems to have nothing to recommend it over the latter.

The unmodified Madelung operation is seldom, if ever, done now. The long disfiguring scar and the extensive subcutaneous area necessarily exposed to the chance of infection were potent factors against it. But its principle is still effective and we believe that this operation, as modified by C. H. Mayo,[11] is the best single operation of the present time for varicose veins. This does not mean that the modified operation can be applied successfully to every case. On the contrary, there are many cases in which mere extirpation of the internal saphenous vein will not give a perfect cure; but it is nevertheless true that such extirpation must be the essential part of any successful operation for the relief of the condition.

As a preliminary step to any operation that has for its object the transferrence of the venous circulation from the superficial to the deep veins, it must be definitely established that there is neither thrombosis of the deep veins nor any marked impediment to the return of the blood through them. A history of a severe preceding phlebitis should arouse our suspicions. Mayo has adopted the diagnostic expedient of having doubtful cases wear an elastic stocking or a bandage from the toes to the knee for a week. If this gives comfort to the patient and relieves the symptoms, it is assumed that the deep veins are sufficiently patent and the following operation is usually done:

The leg is held in an elevated and extended position by means of an ankle-support which is attached to an ordinary, upright, gynæcological standard. This position serves a double purpose; it checks hemorrhage and renders more accessible the field of operation. The internal saphenous is then divided between hæmostats in the upper third of the thigh. The proximal end is ligated; the distal end is threaded into the ring of the enucleator and again grasped with a hæmostat. The vein, put under moderate tension by a pull on the hæmostat, is then extirpated subcutaneously by pushing the enucleator in the direction of the knee. When six to eight inches of the vein has thus been freed, the ring is pushed up against the skin from beneath, a short incision made down on to it, the vein grasped, the enucleator withdrawn, the freed length of vein pulled out through the short incision, rethreaded into the ring, and another length is then extirpated in a similar manner. This process is repeated until the vein has been removed down to a point four to six inches below the knee. The lateral branches are torn as met and usually do not bleed. Occasionally, however, there may be bothersome hemorrhage; this can be controlled by pressure with a gauze pad or by light tamponing of the incision from which it comes. If

the main trunk breaks during the manipulation, pressure is applied over the point of rupture, a short incision is made lower down over the vein, the vessel divided between hæmostats and extirpated from below upwards to the point of rupture. As a substitute for the enucleator, long forceps which form a ring at the end when closed are sometimes used. Occasionally torsion is employed for removing portions of veins, especially when we are dealing with branches. The external saphenous, when varicose, is also extirpated.

At the Carney Hospital my colleague, Dr. Munro, and I have operated on fifty-seven cases of varicose veins in the last three and a half years. The method described above has been approximately the method used in the great majority of cases. Our experience has emphasized several features which, though unessential, are nevertheless worthy of note. We extirpate the internal saphenous to as low a level as possible. Mayo writes that his method is not always applicable, and that in about ten per cent. of the cases either extirpation by the open method or subcutaneous extirpation by some other form of blunt dissection must be substituted for it. We have been led to the conclusion that such substitution must be made in considerably more than ten per cent. of the cases. When the veins are brittle, when they are unusually thin, when the subject is very fat, when the veins are very large and thrombotic, when a considerable degree of periphlebitis is present, or when calcareous deposits have formed, the enucleator does not work well. However, we have yet to see the case in which at some stage of the operation the enucleator has not been of some service. In general, the instrument is more effective in the thigh than below the knee. In a considerable number of cases, especially when the veins are enlarged, thickened and thrombotic, the index finger has proved to be a very efficacious instrument for subcutaneous extirpation.

We have also found it wise, when a branch cannot be torn with the use of moderate force, to make a short incision down on to the enucleator at the point where the branch joins the main trunk, to unthread the latter and, after extirpating the branch as far as can be easily done, to ligate it and then to proceed with the removal of the main vessel. We make a point of ligating the main trunk very close to the saphenous opening and of tying any large branches entering the internal saphenous in the neighborhood.

Any mass of varicose veins, wherever in the lower extremity it may be located, is dissected free and removed. Usually such masses can be removed only through a moderately long incision. Portions of visibly enlarged veins in the leg other than the internal saphenous are also removed, even though at the time they may not show a varicose condition. If there are numerous visible venous channels coursing up the leg, even though the enlargement be only moderate, a partial Schede is done in addition to the usual operation; that is, a "horse-shoe" incision is made, extending perhaps half-way around the leg in the upper third and all superficial veins exposed by the incision are divided between ligatures. All this, of course, takes additional time, but we believe that it is time well spent. Following Mayo's advice, we avoid making incisions in and very close to the fold of the knee, because of the danger of keloid formation in that locality.

All incisions are closed by lock sutures of horse-hair and covered with collodion dressings. It is our practice to close the incision through which the internal saphenous is first tied just as soon as the first length of vein has been extirpated. This lessens the chance of infection. Ulcers are either excised and skin-grafted or thoroughly curetted and allowed to heal by granulation. Mayo[11] describes an ingenious way of dressing these ulcers and grafts so that

their progress may be observed without disturbing the dressing of the whole limb. Eczematous areas are painted with compound tincture of benzoin. Superficial abrasions are wiped with tincture of iodine before proceeding with the operation. Finally the lower extremity is wrapped in gauze, a bandage is applied from the toes to the groin and around the abdomen in the form of a spica; the patient is put to bed with the leg elevated on a pillow. The duration of the stay in bed averages about two weeks. After leaving the hospital the patient usually wears a bandage from toes to knee for three to four months.

The gist of the whole matter is that, while removal of the major portion of the internal saphenous vein is the essential part of the operation, other procedures must be added, suited to the conditions of each case.

Subcutaneous extirpation is by no means a recent operation. Narath[18] gives a most interesting historical account of the various operative procedures for the treatment of varicose veins. Subcutaneous extirpation was first described by Oribasius (325–403 A.D.). It is thus about 1500 years old. It fell into disuse in the later Middle Ages and was revived again after the advent of antisepsis.

The operation is not entirely without danger. Embolism, particularly of the lungs, has followed it several times; Goerlich[10] mentions six cases. Miller[4] describes a probable case occurring in the Johns Hopkins series, and mentions another reported by Sylvester. It has followed both simple and extensive operations, but fortunately is very rare. As yet, we have not met with it at the Carney. The other danger is sepsis. Some degree of it is difficult to avoid because of the presence of eczema or ulceration in many of the cases. We have once seen severe, post-operative, erysipelatous inflammation of the leg. Fortunately it was not fatal, but the patient was seriously ill for some days.

In our series of fifty-seven cases we have done the so-called Mayo operation forty-six times. We know the present condition in twenty-eight of these patients; twenty-four of them are perfectly well; in three the result is fair and can, I think, be made perfect by extirpation of some of the smaller veins which, though not particularly enlarged at the time of operation, have become so in the meantime. The fact that this post-operative enlargement has occurred in these smaller veins has caused us to become more and more radical in our primary operative procedures and to make them more extensive.

Our only really poor result is in a woman who now has a rather marked œdema and more or less constant dull pain in both legs. We believe the failure in this case to be due to the fact that we operated before we were certain that there was no obstruction to the return of the blood in the deep veins; for there can be no doubt that in this case there was and is a partially obstructed deep venous circulation. With our present knowledge operation would not have been advised.

We have done the Trendelenburg operation, modified and unmodified, in twelve instances. Of the six patients whose present condition we know, three are well; three others showed recurrence, two of whom have been cured by the modified Mayo procedure.

We have done the Schede operation twice and excision of portions of the internal saphenous twice. The post-operative history of these cases is unknown to us.

As we have previously stated, recurrences are said to increase in number as the post-operative period lengthens in duration. This applied, however, only to the results of the Trendelenburg and Schede operations. With us so-called recurrences after the Mayo operation, when they have come at all, have appeared within the first six months of the post-operative period. Of course, our work has

covered a period of only three and a-half years, and it may be said that so short a lapse of time does not permit of our speaking of results with definite assurance; but we believe that after the operation as we now practice it a recurrence, as such, can not take place. Some of the remaining branches may enlarge and, perhaps, even become varicose, but re-establishment of the superficial venous circulation is impossible. We feel that in properly selected cases both a subjective and an objective cure may be promised in practically all instances, and that examining surgeons for police and fire boards can now have no sound reason for rejecting as candidates for either the police or fire-departments men who have undergone operation for varicose veins and remain well for a year thereafter.

To sum up in brief:—

1. The important etiological factor is a congenital one; in a small proportion of cases it is inflammatory.

2. The operation that will give the best results must have as its essential feature extirpation of the internal saphenous vein; but to this such other procedures must be added as are suitable to each individual case.

3. Before operative measures are undertaken, it must be demonstrated that the deep venous circulation of the lower extremity is not obstructed to any marked degree.

4. In properly selected cases it is believed that a permanent cure may be promised.

5. The operation is almost but not entirely free from danger.

REFERENCES.

1. Kennedy.—Brit. Med. Jour., Oct. 29, 1904, p. 1151.

2. Bennett.—Wood's Med. and Surg. Monographs, Vol. V, 1890, p. 733.

3. Schambacher.—Deut. Zeit. für Chir., Bd. 53, 1899, p. 575.

4. Miller.—Johns Hopkins Hosp. Bull., Sept., 1906.

5. Trendelenburg.—Beitr. zur klin. Chir., 1890-91, Bd. 7, p. 195.
6. Cited by Georgi.—Deut. Zeit. für Chir., Vol. 73, p. 546.
7. Schede.—Berl. klin. Woch., 1877, No. 7.
8. Madelung.—Verhandl. der deutsch. Gesellsch. für Chir., 1884, p. 114.
9. Ledderhose.—Deut. Zeit. für Chir., 1903-04, Vol. 71, p. 401.
10. Goerlich.—Beit. zur klin. Chir., Bd. 44, p. 278.
11. C. H. Mayo.—Surg., Gyn. and Obstet., July, 1906.
12. Narath.—Deut. Zeit. für Chir., June, 1906, p. 104.

DISCUSSION.

Dr. G. H. MONKS, of Boston: The subject has been so thoroughly presented by Dr. Bottomley that little remains to be added. The number of cases of varicose veins which come to operation is constantly increasing. Whereas years ago comparatively few cases were submitted to operation, at the present time cases of varicose veins form an appreciable proportion of the total number of operations in a general surgical clinic. This, of course, is for the reason that by improved technique, including among other things a more extensive removal of the veins than was formerly practised, surgeons are getting better and better results, and the cases where operation is contraindicated are getting fewer and fewer.

I quite agree with Dr. Bottomley that the best single operation of the three which he has spoken of is the Madelung operation, or one of its modifications; in fact, it has been so satisfactory to me that I have used it exclusively in the 26 cases which I found after a rather hasty search through my records. The other two operations, the Trendelenburg and the Schede, do not appeal to me. I might think differently if I should use them, but, to my mind, the Trendelenburg does not seem radical enough, and the Schede runs too much risk of being followed by unpleasant results caused by division of the veins.

I think it is well to emphasize the importance, on account of its probably lessening the danger of embolism, of working from above downwards rather than in the opposite direction. This method of precedure has also the advantage of enabling the operator to dissect the various branches most effectively.

Dr. Bottomley should be thanked for having brought to the notice of the profession this report which shows how much can be accomplished by means of the latest methods of operating.

Dr. J. B. Blake, of Boston: There are two aspects of the operation for varicose veins, which are worthy of a few words. Although the operation seems to be one of the simplest in surgery, its field being superficial to the deep fascia, it is nevertheless not without its dangers. Of these sepsis is the most important, and it may be so severe as to not only ruin the operative result, but also endanger the patient's life. Simple though the operation is, the patient's general condition must be considered, just as thoroughly as the local lesion. Failure to include this element of risk was, I believe, the determining factor in the following case, the only fatal outcome of an operation for the relief of varicose veins that is recorded in Boston. The patient was an old feeble woman, operated on by a young doctor who had recently finished the duties of surgical house officer in a Boston Hospital. There was an open ulcer, as well as an extreme degree of varicosity, threatening rupture and hemorrhage. The operation was a local dissection. Immediate phlebitis supervened; the patient was sent to a hospital, where in spite of every effort, toxæmia and death followed within a week. Aseptic precautions were taken at the time of operation as a matter of course, but the patient was operated on in her own home. A longer preparation, increased attention to improving her general condition, would undoubtedly have averted this disaster. It seems worth while drawing attention to this single case, as at present every one seems to be doing surgery, at all times, under all conditions and upon all sorts of surgical risks. Necessarily, some patients suffer, and from causes that are not legitimately essential to modern surgery.

The other aspect of the operation is the precise reverse of the foregoing: in patients whose general condition is satisfactory, but who have large, and slowly healing chronic ulcers, it is not always necessary to postpone the operation until these ulcers heal, even though a considerable portion of the operative field lies in immediate proximity to the ulcer. If unusual care is taken in cleansing the stain, and the ulcer

be entirely and permanently shut off from the rest of the leg by a special dressing, applied before the anæsthetic is administered, and later covered by the sterile sheets, the operation may proceed to within one cm. of this dressing with perfect safety. This is of course intended to apply only to patients in hospitals, or under ideal home conditions, but both Drs. Cotton and Blake have had several such cases in the past two years, in which the operative result was as good as if no ulcer was present. As a rule, the ulcer heals more rapidly after the operation.

ARTICLE XXX.

THE TREATMENT OF TYPHOID FEVER WITH SPECIFIC SERA, FILTRATES AND RESIDUES (Vaughan).

By MARK W. RICHARDSON, M.D.
OF BOSTON.

READ JUNE 11, 1907.

THE TREATMENT OF TYPHOID FEVER WITH SPECIFIC SERA, FILTRATES AND RESIDUES (VAUGHAN).

IT is now eight years since the writer first took up the study of immunity in typhoid fever with the avowed purpose of developing, if possible, an effective, specific method of treatment for the disease.

Financially the study has been supported by two grants of the Dalton Scholarship at the Massachusetts General Hospital.

For clinical material I am indebted for all but two cases to members of the Medical Staff at the same institution, and especially to Doctors Shattuck, Cutler and Minot. One case was referred to me by Dr. Arthur Broughton of Jamaica Plain, and another by Dr. H. V. Reynolds of Dorchester.

Invaluable aid in the production of a specific serum was furnished by the State Board of Health, which put at my disposal its stable for the care and inoculation of horses. Finally to Professor Theobald Smith I desire to express my deep appreciation of his continued interest and encouragement.

From unavoidable circumstances the work has been carried on spasmodically, and is in many ways incomplete. It has been pursued along the broadest of lines. The finer problems of immunity have been left untouched. Briefly expressed, the effort has been made first, by inoculating the horse with typhoid material, to produce a serum which

should be of assistance in human typhoid infection; and in the second place by inoculation of the typhoid patient himself with small doses of typhoid material to hasten his own immunity and cure. The latter method is, of course, similar to that of specific inoculation introduced recently by Sir A. E. Wright for staphylococcus and other bacterial infections. Thus far no attempt has been made to control treatment by observation of the so-called "opsonic index" of the blood.

This study covers observations upon 130 cases of typhoid fever, treated (in addition to the routine hospital treatment) with the following specific fluids. Anti-typhoid serum was used in 35 cases, and was produced by inoculating horses over a period of one to two years with gradually increasing doses of dead typhoid bouillon cultures. The serum of the horses was then given to typhoid patients, (*a*) subcutaneously (5 cases in daily doses of 10 to 100 cc.); (*b*) by mouth or rectum or both (18 cases—dose 100 to 1000 cc. daily); (*c*) in pill form with special enteric coating (7 cases); (*d*) 5 cases subcutaneously with equal amounts of normal serum (15 cc. of each daily), to secure assistance of any complements which might be present in the normal serum. It was hoped that serum given thus in unusual method and dose might produce better results than had been previously obtained.

In a second series, 74 in number, a typhoid filtrate was used, always subcutaneously. This filtrate was obtained by passing living bouillon cultures through porcelain filters. The age of the cultures varied from two days to six weeks. The results obtained seemed to be the same whatever the age of the culture. The dose averaged 3 cc. daily.

In a third series 21 cases were treated with "non-toxic residue" of the typhoid bacillus as prepared by Professor Victor C. Vaughan of Ann Arbor, and kindly sent to me

by him (daily subcutaneous dose 2-10 cc.). This residue, though non-poisonous, contains all the essential immunizing portions of the typhoid organism.

Finally, in a series of 28 cases (6 of which are already included in series 3) it was attempted to prevent relapses in typhoid fever by the continued inoculation of the non-toxic residue at increasing intervals during convalescence.

Now, in any consideration of specific therapy in typhoid fever it is important to bear in mind that we have to do with a problem involving not the simple neutralization of a toxin by an antitoxin, as in diphtheria, but having for its object the increased destruction of the invading germ. This destruction follows the interaction of factors much more complex than those seen in diphtheritic infection, and, furthermore, means the setting free of whatever intracellular toxin those germs contain. Treatment along these lines, therefore, would not be appropriate for patients already severely intoxicated, but should, on the contrary, be inaugurated at the earliest possible stage of the disease.

With our serum, then, we have tried to introduce into the patient's body specific substances destructive to the typhoid bacillus, substances produced by inoculation in the blood and tissues of the horse. In other words, any immunity induced by this treatment is of the passive variety. With the filtrates and residues our object has been to stimulate the patient's own blood and tissues to greater production of specific antibodies, and the consequent shortening of his disease. That is to say, we have tried to hasten and strengthen the active immunity natural to the disease by the introduction into the body of additional small doses of specific infectious material.

That these inoculations, therefore, should be effective and advisable from a therapeutic standpoint it is essential first, that the patient be not so sick that he cannot react with greater efforts towards destruction of invading germs,

and second, that he can stand the temporary increased intoxication due to that destruction.

A comparative study of 130 cases of typhoid fever appears in the following tables, which offer many points of interest, but which must be scanned very critically lest they convey false impressions.

TABLE I.

	Routine Series.	Serum Series.	Filtrate Series.	Residue Series.
No. of cases treated	74	35	74	21
Cases 15 years of age or younger	19	6	16	6
Average day of disease on entrance . . .	12.05	10.4	10.21	11.4
Average day of beginning treatment . .	12.05	15.6	13.83	13.05
Treatment delayed, lack of diagnosis . . .	(20)	8	9	5
Average necessary delay	(6)	4.25	8.88	5
Average length of fever in original disease .	67 cases 27.35	33 cases 30.3	63 cases 27.45	19 cases 32.5
No. of days in hospital .	67 cases 31.77	33 cases 45.75	63 cases 41.57	19 cases 42.42

In the first place the percentage of children in any typhoid series is important because, as is well known, the disease in children is apt to be mild in type under natural conditions.

(2) Typhoid patients rarely come under any form of treatment until the disease has been in progress several days. Hospital experience, as shown by 204 cases, makes

the average day of entrance the eleventh day. This early period, moreover, most important for the inauguration of any specific treatment, is still further lengthened by the fact that, even at this stage, a positive diagnosis is often impossible.

(3) In 204 cases specific treatment could not have been begun immediately in 42 instances, and the average increased delay from that cause would have been five days. Until, therefore, earlier diagnosis becomes available, specific treatment must be severely handicapped. Table II would

TABLE II.

	Day of beginning treatment.	Routine Treatment Series.	Serum Series.	Filtrate Series.	Residue Series.
Average length of fever subsequent to the beginning of treatment.	1–10 day	38 cases 16.55 days	9 cases 15.8 days	19 cases 11.15 days	8 cases 19.37 days
	11–20 day	22 cases 17.26 days	17 cases 16.1 days	34 cases 13.64 days	8 cases 20.75 days
	21–30 day	7 cases 12 days	4 cases 10.5 days	5 cases 18.6 days	2 cases 9 days
	31–40 day	o o	2 cases 9 days	1 case 12 days	o o
	41–50 day	o o	2 cases 7 days	1 case 17 days	o o

seem to bear out this contention, for in spite of delayed diagnosis and treatment, there was in the early cases an apparent shortening of the original fever in the serum and filtrate series.

In the residue series, however, the subsequent fever was apparently lengthened, and statistics thus strengthen what

was my strong impression while the cases were under observation, namely, that these cases were drawn out, but on the other hand seemed unusually mild in character. The coincidence of increased length with increased mildness may be only apparent, but further experience may give it great importance. As to the average stay in the hospital, the cases under specific treatment staid considerably longer,—due undoubtedly to the large percentage in those cases of relapses. One serum case, for instance, went through five relapses and was in the hospital 116 days.

TABLE III.

	Routine Series. 74 Cases.	Serum Series. 35 Cases.	Filtrate Series. 74 Cases.	Residue Series. 21 Cases.
Relapses { True	6= 8 %	8=22 %	9=12.1 %	3=14.2 %
Relapses { Intercurrent	4= 5+ %	3= 8.5 %	4= 5.4 %	1= 4.7 %
Otitis Media	4= 5 %	2= 5.7 %	1= 1.3 %	0= 0 %
Pneumonia	1= 1.3 %	0= 0 %	2= 2.7 %	0= 0 %
Empyæma	0= 0 %	1= 2.8 %	0= 0 %	0= 0 %
Phlebitis	3= 4 %	1= 2.8 %	3= 4 %	1= 4.7 %
Pulmonary Embolism	1= 1.3 %	0= 0 %	1= 1.3 %	0= 0 %
Periostitis	0= 0 %	0= 0 %	1= 1.3 %	1= 4.7 %
Furunculosis	0= 0 %	6=17+ %	6= 8 %	3=14.2 %
Bed Sores	1= 1.3 %	1= 2.8 %	2= 2.7 %	0= 0 %
Abscess Parotid	1= 1.3 %	0= 0 %	0= 0 %	0= 0 %
Empyæma Gall Bladder	1= 1.3 %	0= 0 %	0= 0 %	0= 0 %
Diarrhœa	33=44 %	20=57 %	22=29 %	5=23 %
Typanites	15=20 %	11=31 %	18=24 %	2= 9 %
Hæmorrhage	6= 8.7 %	7=20 %	9=12.1 %	3=14 %
Perforation	2= 2.7 %	1= 2.8 %	3= 4 %	0= 0 %
Delirium	16=21 %	11=31 %	15=20 %	6=20 %
Severe Renal Disturbance	9=12.1 %	8=22 %	7= 9 %	2= 9 %

The comparative frequency of complications appears in Table III.

Taken with the great reserve with which all such tables must be observed, we see that the impression of mildness in the residue series is further borne out.

The high percentage of diarrhœa in the serum cases was due undoubtedly to the rectal administration of the remedy, and consequent irritability of the lower bowel.

The frequency of hemorrhage was largest (4) in the cases treated with combined normal and typhoid serum subcutaneously. In one case only was it of serious import, being followed later by perforation and death. This high percentage was probably simply a coincidence.

Under the head of severe renal disturbance was included all cases showing more albumen and a greater number of casts than could be expected from ordinary febrile conditions. The serum series shows apparently a very high percentage of kidney involvement, but close scrutiny shows that four of the eight cases had this urinary condition before specific treatment was begun.

In studying the table of mortality (Table IV) one is struck immediately, of course, by the large mortality in the series of seventy-four filtrate cases as compared with seventy-four routine cases. This very unfavorable showing is very misleading, for in the first place the death from embolism occurred without warning nine days after inoculation had ceased, and when the patient was apparently becoming rapidly convalescent. There was a similar case of embolism in the untreated series.

The case which died of pneumonia had practically finished a mild typhoid infection when double otitis media required etherization. Pneumonia and death supervened in four days.

A perforated appendix claimed a third case. Treatment was not begun till the thirty-second day. This case had

TABLE IV.

	No. of Cases.	No. of Deaths.	% of Deaths.	Cause of Death.	Remarks.
Routine Treatment Series	74	7	9.4	Toxæmia	
				"	
				"	
				Hæmorrhage	
				Hæmorrhage and Perforation	
				Toxæmia	
				"	Embolism and Pneumonia
Serum Series	35	2	5.7%	Toxæmia	
				Hæmorrhage and Perforation	Operation
Filtrate Series	74	11	14.8%	Pulmonary Embolism	
				Hæmorrhage	
				Pneumonia	Otitis Media
				Perforated Appendix	
				Toxæmia	
				"	
				"	
				Hæmorrhage	
				Perforation	Operation
				Hæmorrhage	
				Perforation	Inflammation of both Maxillary Joints
Residue Series	21	2	9.5%	Hæmorrhage	Mixed Infection with Staphylococcus Aureus
				"	

been very severe with hemorrhage on the twenty-fifth day. The case was doing well when perforation demanded immediate operation. A case of perforation in the routine series, on the other hand, came to operation and recovered, a fact fortunate for the individual, but very disturbing in a consideration of mortality statistics.

Finally, it is very important to know that all fatal cases, with one exception (second perforation), were severely sick when they came under treatment, and the outlook in such cases, as we have seen, is not encouraging.

In the residue series the first fatal case, as proven during life and autopsy, was a mixed infection with the staphylococcus aureus. With our present knowledge this case would have received a mixed treatment of typhoid and staphylococcus vaccines, and might possibly have been saved.

Hemorrhage in typhoid, which claims so many victims, seems to be due in part, at least, to a faulty coagulability of the blood. It is not probable that anti-typhoid measures alone can materially affect mortality from this cause.

In this investigation certain phenomena have been observed which deserve mention.

In the serum series one case alone was marked by an unusual manifestation. A woman, moderately sick, had received subcutaneously for six days 100 cc. of antityphoid serum. On the seventh day, coincident with a very marked urticaria, the temperature fell suddenly, and her pulse became very weak. She was in a dangerous condition for twenty-four hours. The circulation then rapidly improved, in fact the clinical condition was better than before the occurrence. I ascribe it either to a sudden destruction of many typhoid bacilli with resulting hyperintoxication, or to some internal manifestation allied to the urticaria, and due to the same cause, namely large amount of horse serum.

In the filtrate series, twenty-nine cases were remarkable through the occurrence of quite characteristic reactions,

namely, a chill coming almost always one hour after inoculation, and accompanied by a sharp rise in temperature, pulse and respiration. This increase in symptoms was followed in many instances by a marked decrease in the temperature and the pulse, and considerable improvement in the general condition of the patient. (See Chart 1.) After a short interval the temperature in most cases went up again, but in a few instances the temperature remained at normal permanently. (See Chart 2.)

This phenomenon, though rarely, is seen in the natural course of typhoid. (See Chart 3.) Tentatively I would explain it by supposing that ordinarily the destruction of typhoid bacilli with resulting toxin production takes place quite continuously, and sudden fluctuations in the intoxication are rare. Occasionally, however, under natural conditions, and especially under filtrate inoculation, destructive agencies are brought into sudden and excessive action with the resulting temporary increase in symptoms. This excess of action, as I take it, might well be followed temporarily by no action at all, and that would explain the fall in temperature and pulse. After a short interval the destructive apparatus would again take up its work, and the intoxication would go on (Chart 1) unless all the germs had been killed. In this case the temperature would remain normal, there being no further bacilli to furnish intracellular toxin. (Chart 2.) It may be possible to prove the truth or falsity of this theory by observation on the opsonic index before and after chill.

Another filtrate case was interesting because it shows very probably what might be accomplished in a majority of typhoid cases if they could but receive specific treatment very early in the disease. (See Chart 4.) This patient came in very mildly sick on the 10th day of the disease. Two inoculations with filtrates produced temporarily a marked increase in his symptoms, but this was followed

immediately by a rapid and permanent convalescence. That these filtrates, furthermore, are any thing but indifferent fluids I demonstrated on myself by a subcutaneous inoculation of 2 cubic centimeters. For twenty-four hours I had fever, headache, backache, increased pulse rate, in fact all the common symptoms of an acute infection. Moreover, in three weeks my blood developed a faint but perfectly distinct typhoid reaction.

Finally, I have to record a series of twenty-eight cases in which it was attempted to prevent relapses in typhoid. To accomplish this the patients were inoculated at increasing intervals during the convalescence with small doses (generally 2 cc.) of Vaughan's non-toxic residue.

In twenty-eight cases there was one relapse. The incidence of relapse among the untreated cases during the same epidemic was twenty-five in one hundred and thirteen cases. That is to say, the percentage of relapses was reduced apparently from 22.1% to 3.57%. My strong impression that this gratifying result is real is reinforced by the behavior of some of the cases under inoculation.

For instance, in Chart 5 it is seen that four inoculations had been given during the convalescence without clinical manifestations. Five days after the last inoculation, however, the temperature began to go up in a very suspicious manner. An inoculation exactly similar to the previous ones now produced a very sharp reaction with a chill. The next morning, however, the temperature returned to normal and remained there. I believe this case would have relapsed if the beginning re-infection had not been nipped in the bud by the inoculation.

SUMMARY.

The study of specific therapy in one hundred and thirty cases of typhoid fever has brought out the following facts:—

(1) That specific therapy, confined to the original disease, increases, apparently, the tendency of the disease to relapse.

(2) That by therapeutic inoculation, however (Vaughan's non-toxic residue) continued into convalescence, the relapse in typhoid fever can be largely eliminated. This claim is made with much assurance, but is, of course, subject to confirmation by observation upon a much larger series of cases. If relapses can be prevented then it follows almost certainly that mild typhoid infections can be aborted. This study would show, furthermore,

(3) That to get the best results cases must be seen as early as possible in the infection, but

(4) That an early diagnosis in typhoid is oftentimes impossible, and, that, for this reason, specific therapy suffers under a severe handicap.

(5) That antityphoid serum was no more effective than filtrates and residues and was infinitely more expensive.

(6) That typhoid filtrates may exert a powerful effect upon the clinical course of the typhoid process, their use being followed in many instances by chills, or rise in temperature and pulse, these being followed in their turn, oftentimes, by marked fall in temperature, pulse and respiration, and a general improvement in the clinical picture. The nature of these reactions is still unknown. It is hoped that further study, especially that of the opsonic index, in connection with these reactions, may give valuable information concerning their nature.

(7) That the non-toxic residue of the typhoid bacillus as prepared by Professor Vaughan seems to make the typhoid process longer, but milder. As stated above, it is apparently very effective in the prevention of relapses.

CHART 1.

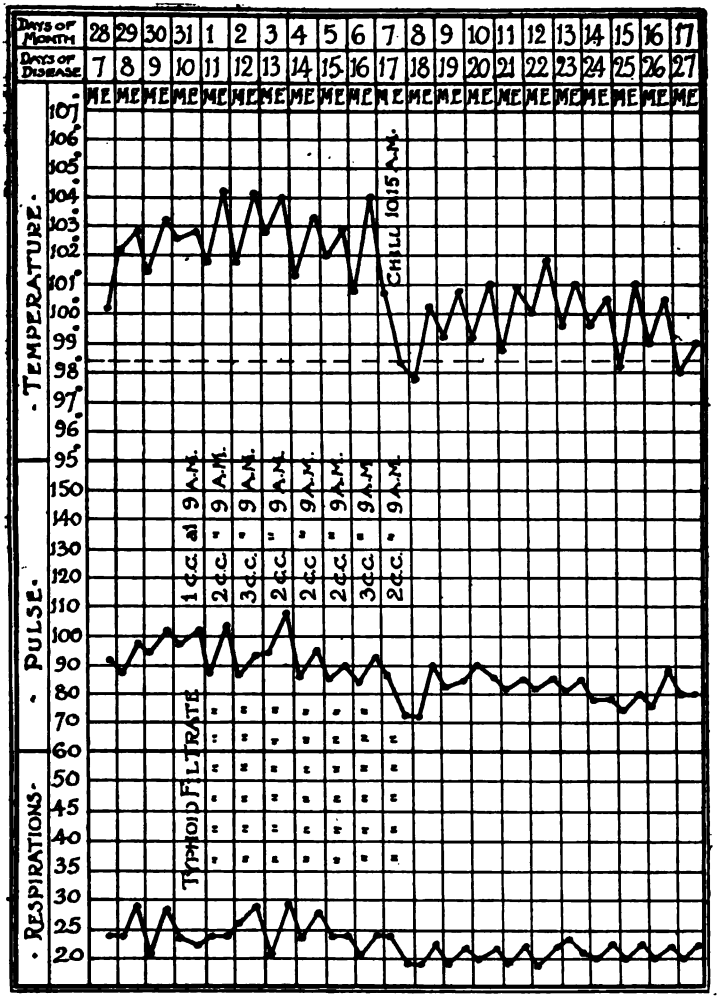

Days of Month
28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17
Days of Disease
7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27
M E
· Temperature ·
· Pulse ·
· Respirations ·
Chill 10.15 A.M.
Typhoid Filtrate
1 c.c. at 9 A.M.
2 c.c. " 9 A.M.
3 c.c. " 9 A.M.
2 c.c. " 9 A.M.
2 c.c. " 9 A.M.
2 c.c. " 9 A.M.
3 c.c. " 9 A.M.
2 c.c. " 9 A.M.

CHART 2.

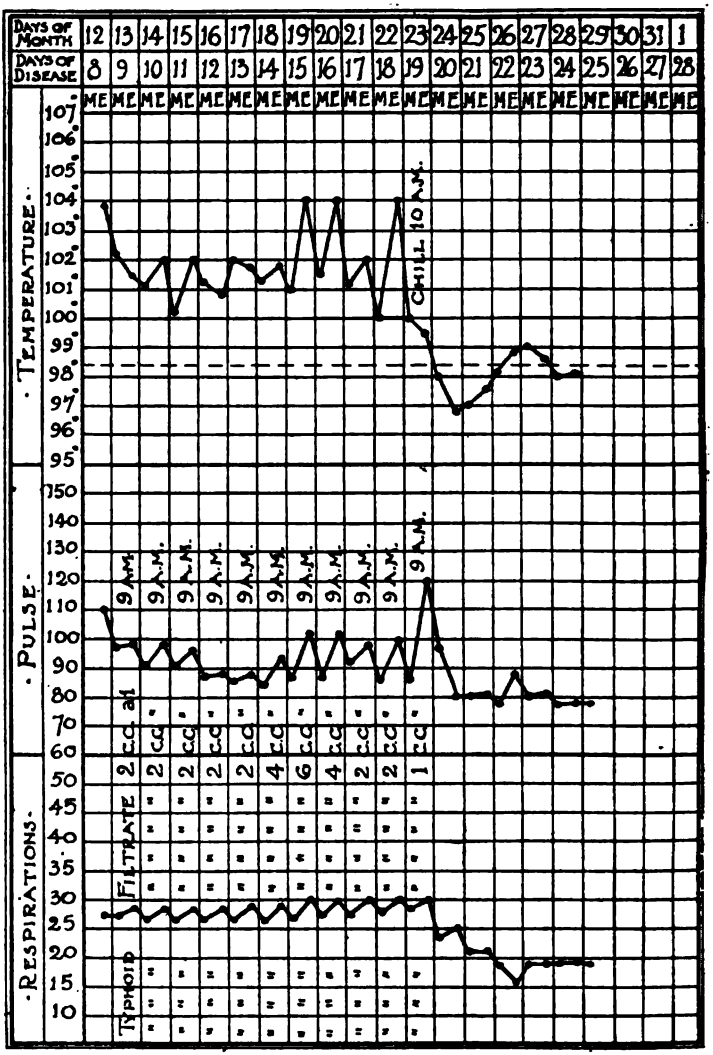

Days of Month
Days of Disease
Temperature
Pulse
Respirations
Typhoid
Filtrate
9 A.M.
Chill 10 A.M.

CHART 3.

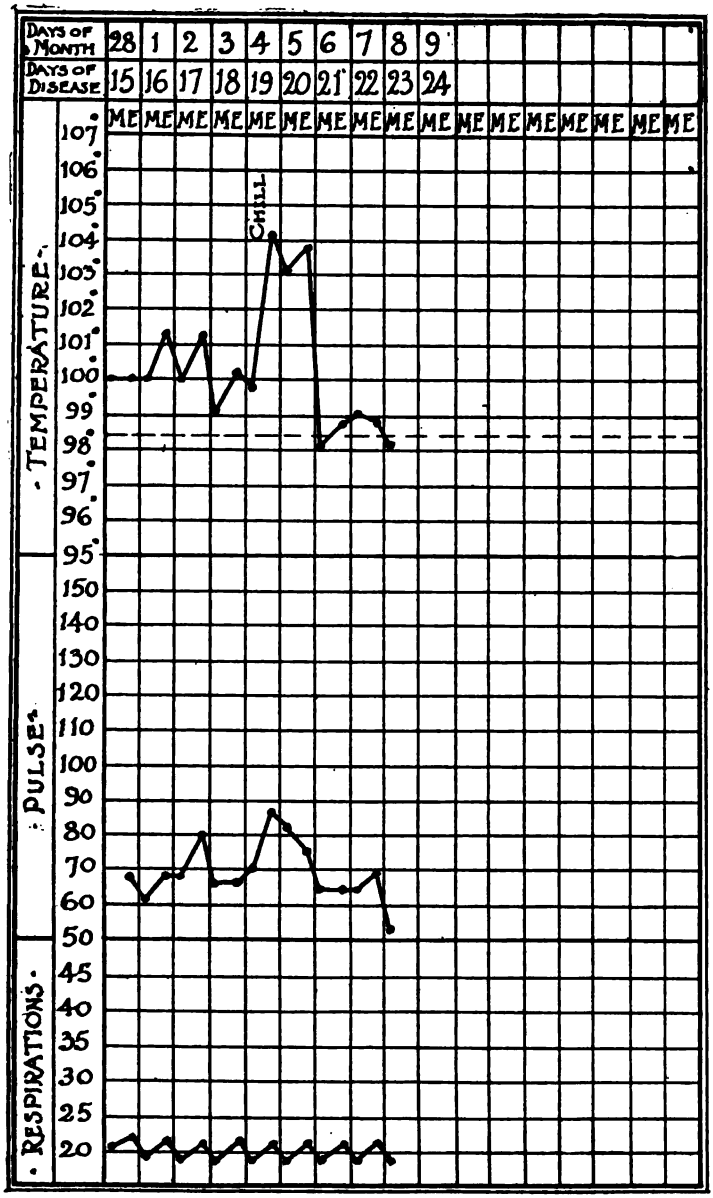

Days of Month
28 1 2 3 4 5 6 7 8 9
Days of Disease
15 16 17 18 19 20 21 22 23 24
ME
Chill
Temperature
Pulse
Respirations
107
106
105
104
103
102
101
100
99
98
97
96
95
150
140
130
120
110
100
90
80
70
60
50
45
40
35
30
25
20

CHART 4.

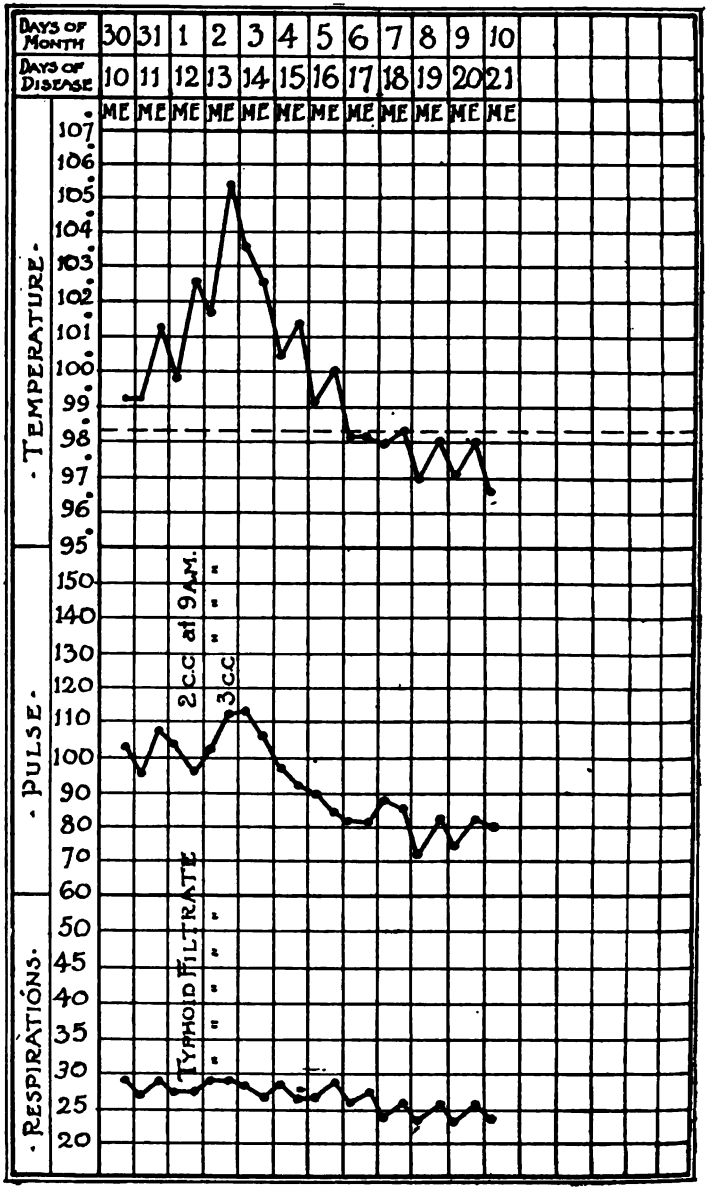
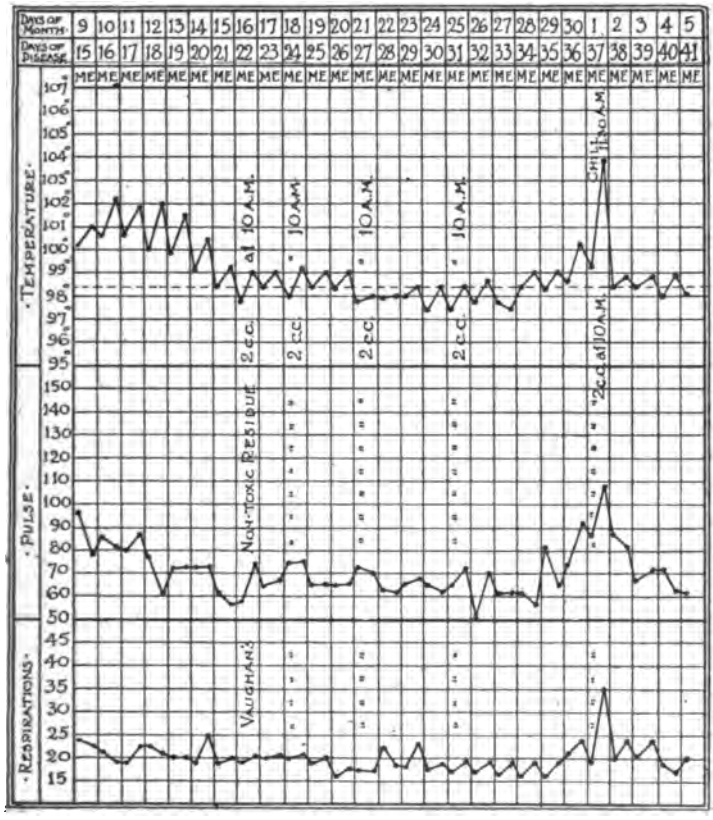

Days of Month
30 31 1 2 3 4 5 6 7 8 9 10
Days of Disease
10 11 12 13 14 15 16 17 18 19 20 21
ME
· Temperature ·
107 106 105 104 103 102 101 100 99 98 97 96 95
· Pulse ·
150 140 130 120 110 100 90 80 70 60
2 c.c. at 9 A.M.
3 c.c.
· Respirations ·
50 45 40 35 30 25 20
Typhoid Filtrate

CHART 5.


Days of Month
Days of Disease
TEMPERATURE
PULSE
RESPIRATIONS
Non-Toxic Residue
Vaughan's
2 c.c. at 10 A.M.
Chill 11.0 A.M.

ARTICLE XXXI.

EXPERIENCE WITH OPSONINS AND BACTERIAL VACCINES IN THE TREATMENT OF TUBERCULOUS AND NON-TUBERCULOUS ARTHRITIS.

By CHARLES F. PAINTER, M.D.
OF BOSTON.

READ JUNE 11, 1907.

EXPERIENCE WITH OPSONINS AND BACTERIAL VACCINES IN THE TREATMENT OF TUBERCULOUS AND NON-TUBERCULOUS ARTHRITIS.

OBSERVERS of new phenomena lay themselves open to errors both of observation and interpretation. Particularly is this true when the phenomena concern problems, the solution of which has been sought long and diligently. There has never been a time when so many well trained men were working upon the problems of physiological chemistry and bacteriology as there are at the present day. At this time also, great popular as well as professional and scientific interest has been aroused in the subject of tuberculosis, and to a lesser degree in chronic non-tuberculous joint disease. Witness the money being advanced for the anti-tuberculosis crusade, and the attention devoted by all classes of society to attempts made to wipe out this curse. In the midst of all this popular enthusiasm and highly specialized scientific attainment, has come a new method which has much to recommend it from the theoretical and laboratory points of view. The questions at issue are, Do the clinical results correspond with the laboratory findings? Are the limits of error in methods of laboratory investigation so wide that the conclusions which seem legitimate from such observations may be erroneous? To the first of these questions only will the writer devote his attention.

The literature on opsonins and bacterial vaccines is getting to be quite voluminous, but the greater part of it is concerned with laboratory technique and theoretical con-

siderations of the processes by which immunity may be brought about. A very few reports of clinical experience are on record, and many of these are valueless because of the paucity of data recorded, or because of the obvious inaccuracies of the observations.

Clinical experience must be the court of last resort in matters of this sort for many years to come. The human being is not a chemical retort into which reagents may be injected and results obtained which are identical with those we look for when experimenting in the laboratory upon the lower orders of animal life. Wonderful as have been the advances made, rational conservatism must be the prevailing state of mind of those who would not be carried away by their enthusiasm.

Attempting to put ourselves in this mental attitude, it is proper to review the facts. The laboratory side of the question has been thoroughly presented in the previous paper, and even though clinical experience cannot wholly bear out the brilliant results obtained along purely scientific lines, this does not in any way vitiate the results of such enquiry.

Successes obtained by some observers in the treatment of septic wounds, boils, acne, and similar localized infections, encourage the clinician to hope that there are as yet some ill-understood obstacles in the way of the application of the method to those conditions where it has failed. The good results in some other fields, notably in the treatment of diphtheria, by an analogous method, also offer much encouragement. One cannot read the literature covering the clinical experience of the writers without considerable misgiving as to the practical value of bacterial vaccination. Neither can he be particularly encouraged in the hope that the opsonic index is to be of advantage as a guide to the dosage or the repetition of inoculation. The consideration of its value as a diagnostic aid has not been extensively con-

sidered in the literature. Its value from this point of view, in the writer's experience, will appear in the course of this paper.

A few citations from current literature to show the character of the reports of clinical observers may not be amiss. These are cited because in most cases the writer believes acceptance of the findings at their face value would lead the uninitiated reader to improper conclusions regarding the efficacy of the method. French*, in the British Medical Journal, reports a case of tuberculous dactylitis and tuberculous disease of the os calcis, both having fistulæ. The child had a low opsonic index to the tubercle bacillus. 1/5000 mgr. of tuberculin "R" was given. A negative phase lasting nearly four weeks was the result. A second inoculation, again followed by a prolonged negative phase, was accompanied by some improvement. A third inoculation was given and the sinuses then promptly healed, and at the end of four months the writer says the patient was practically well. Such a case as this, even though it represents an observer's personal experience with bacterial vaccines in the treatment of tuberculous arthritis, should not be given much weight in determining the value of tuberculin in the treatment of bone disease.

Ohlmacher,† writing in the Journal of the American Medical Association, reports a remarkable series of cases of various sorts treated by bacterial vaccines. In determining the dosage and frequency of inoculation he was admittedly lax in the observation of the opsonic index, without which Wright himself, and Ross, the principal exponent of his technique on this side of the water, claim satisfactory results cannot be obtained. In spite of this failure the results were so uniformly good in all cases, he concludes, that, with the possible exception of diphtheria antitoxine

*H. French.—Brit. Med. Jour., Feb. 2, 1907, page 256.

†Ohlmacher.—Journal Am. Med. Assoc., Feb. 16, 1907.

in the treatment of diphtheria, there is no more valuable reagent than bacterial vaccines for the treatment of many surgical infections.

Kellogg* finds the determination of the opsonic index of value diagnostically in the following reported case. A young woman consulted him because of a troublesome hip. There had been an injury to this joint some years previously, and she had been obliged to use crutches during all this time. The head of the affected femur was dislocated. *One* observation showed the tubercular opsonic index to be .22. From this he concludes that the disease was a tuberculous one. In all probability this was true, but to have argued that it was because of *one* positive result in testing the phagocytic power of her leucocytes, constitutes a very insecure basis for such a conclusion. A second case is reported by the same author. A patient came under his care for a pleural effusion. The chest was aspirated, but guinea pigs inoculated with this fluid showed no signs of tuberculosis. A diagnostic dose of tuberculin was given and a typical reaction followed. Between July 12th and August 24th, of the same year, she was inoculated with Koch's tuberculin, "R," not over three times (the record states at ten day intervals). At the end of this period her physical signs had all cleared up and her weight had increased 7½ lbs. I submit that the proof of the tuberculous character of her effusion is lacking, and that to offer such a case as a striking example of the curative value of tuberculin in such conditions is improper.

Wolff† reports some interesting results in the treatment of pneumonia by a pneumococcus vaccine. He feels that raising the opsonic power of the patient's serum in pneumonic cases was attended by more favorable results than attends the usual treatment of pneumonia patients, but thinks

*Kellogg.—Northwest Med. Jour., Vol. V, No. 1, page 20.

†Wolff.—Jour. of Infectious Diseases, Oct. 30, 1906.

too few observations have been made to justify any positive assertion that such is the case.

Hollister* reports several cases too briefly to be of any scientific value. The general impression conveyed was that there was improvement, but not such as would be inconsistent with other and general treatment, simultaneously carried out.

Ross,† in a recent article, reports success in a number of cases of lupus, empyema, staphylococcus infection, endocarditis, and Malta fever.

Bunch‡ refers to two cases where bacterial vaccines were employed to reinforce surgical treatment. He believes that this method offers great possibilities in the management of surgical tuberculosis by diminishing the negative phase produced by operating upon tuberculous foci, when the resistance of the patient has not been properly fortified. He also advocates the use of vaccines after operation during long periods, for the same reason. He cites a case of extensive lupus operated upon by Mr. Godlee. The operation was an excision of the lupoid tissue, followed by skin graft. Before the operation and for some time after, injections of tuberculin were carried out, in consequence of which he believes, that the entire graft was successful. 1-1000 mgr. were used at ten day intervals. He also reports a case of acetabular hip disease which had been treated for 1½ years, by apparatus and bed treatment, without any appreciable progress. An abscess developed, for which operation was performed. Just before operation 1-1500 mgr. of tuberculin was injected, and this was repeated during the succeeding four months, at the end of which time the patient was reported as practically well. He also refers to an article of Turton's, in which thirty out

*Hollister.—Surgery, Gynæcology, and Obstetrics, Vol. 3, No. 6, p. 740.
†Ross.—The Canada Lancet, Jan. 1907.
‡Bunch.—Lancet, London, Jan. 19, 1907, page 145.

of thirty-four cases of different sorts were treated with bacterial vaccines with very satisfactory results.

Fraser* reports 41 observations upon the opsonic power (tubercular) of the blood in healthy individuals. In the first place she attempted to establish a mean of variation. In the second place she compiled a table of 43 indices from individuals who were suffering from other diseases than tuberculosis. She also reports observations upon the tuberculo-opsonic index in nine members of tuberculous families. Her next series of observations was upon 91 cases of tuberculous disease. She found no striking diagnostic value in the tuberculo-opsonic index. In 58 of these cases the index was higher than that seen in health, while in 33 it was within normal limits. She found she could not classify her cases according to their indices as Wright claims to be able to do. The low index did not necessarily show a localized focus; neither did a high index, within the limits of variation, indicate a generalized process.

Brooke† refers to Bullock's remarkable series of staphylococcus infections reported in the Practitioner for Nov. 1905. Eleven cases treated with vaccines showed nine prompt and complete cures.

There is a notable absence of clinical reports in German and French literature. This absence should have considerable more significance than does the too sanguine position of the observers to whom reference has been made. A few good results have been reported following the use of Marmorek's anti-tuberculous serum, conspicuous among which are the cases cited by Hoffa in a recent article.‡

In July, 1906, at the commencement of a four months' service at the House of the Good Samaritan, I began the treatment of tuberculous bone disease with Koch's new

*Fraser.—Glasgow Med. Jour., March 1907, page 194.

†Brooke.—Dublin Jour. Med. Sciences, Sept. 1, 1906.

‡Hoffa.—Berliner klin. Wochenschr. XLIII, No. 8.

tuberculin. The inoculations have been kept up by my colleagues, Dr. Augustus Thorndike and Dr. J. S. Stone, during their services up to the present time. The patients upon whom this treatment had been tried were kept in the Hospital in nearly every instance throughout this entire period, so that in addition to any benefit which may have accrued from these injections, they have had the benefit of sanitarium treatment. They were all advanced cases when treatment was begun and were all children. In addition to these children I have followed the same treatment in a few cases of tuberculous bone disease in the adult, at the Carney Hospital. These patients had not the benefit of as good hygienic treatment as had the group at the Samaritan. It was not possible to keep them in the hospital for as long periods and they could not be controlled as well, because they were adults. They were all bad cases. The laboratory work in connection with these observations was done through the courtesy of Dr. Leary at the Pathological Laboratory of the Tufts Medical School. In private practice I have also treated a few patients with bone tuberculosis in this way and in both private practice and at the Carney Hospital Clinic we have treated several cases of chronic polyarthritis, of the rheumatoid type.

In brief detail the cases and results have been as follows (their charts have been shown in Dr. Leary's paper):

Case I. Mrs. B. 30 yrs. Tuberculosis of the lumbar spine, the left ankle, and the right great toe. Clinical diagnosis confirmed, so far as the ankle lesion was concerned, by histological examination of material removed at operation and by the tuberculous opsonic index. Patient had been under apparatus treatment for the Pott's lesion for two years and for the ankle one year. Ten injections of tuberculin were given, beginning with 1–1000 mgr. and going as high as 1–500 mgr. on two occasions. The Pott's lesion has made good progress; the ankle lesion was much worse than at any time for a period of three months, but

has slowly been improving during the past four months; the lesion on the great toe has caused no trouble at any time during or immediately before the commencement of the injections. Rest and fixation have been enjoined throughout this entire period. The patient has gained in weight, improved in color and general condition, but no more so than has often been experienced in the same time and under the same conditions of hygiene and protective treatment.

Case II. Mr. K. 33 yrs. Tuberculosis of mesenteric and retroperitoneal glands. Suppuration and mixed infection with the bacillus pyocyaneus. There is in all probability at this time an osseous involvement of the lumbar spine. There has been no pathological confirmation of the clinical diagnosis. The case has been under treatment for ten months and has had thirteen inoculations with tuberculin and a streptococcus and pyocyaneus vaccine. During the first five months of treatment there was considerable improvement in general condition and local signs, together with an almost complete closure of the sinus. The past six months his general condition has failed markedly; he has developed a very well defined phthisis, his sputum being loaded with tubercle bacilli, and the local signs in his spine have become much worse. His sinus is discharging very freely and death seems imminent at any time.

Case III. Mr. A. 29 yrs. Tuberculosis of left hip. Excision. Clinical diagnosis confirmed by histological examination. A very acute case of hip disease, said to be of only four months duration at the time of admission to the Carney for excision of the head of the femur. Inoculation commenced before operation in the hope that his local and constitutional resistance might be built up. (Vide opsonic chart.) After operation there was a pyocyaneus infection and he was given pyocyaneus vaccine in addition to tuberculin. He was operated upon Oct. 17, 1906, and discharged Jan. 21, 1907. He improved slowly but reacted, it seemed, better than most cases of such severity, and since he left the hospital is reported to be doing well.

Case IV. Miss M. 48 yrs. Tuberculous cervical adenitis. Phthisis. Chronic infectious polyarthritis. Glands in the neck suppurated. No tubercle bacilli found in smears

from this pus; pus not injected into guinea pig. Tubercle bacilli in sputum. Patient had three inoculations with Koch's old tuberculin, and later this was changed to the new "T. R." There was no appreciable improvement in the local condition of the glands, and her pulmonary process became worse. She had five inoculations of the new tuberculin at ten day intervals. She was discharged from the hospital after a stay of eight months, in a worse condition than at entrance.

CASE V. M. W. 5 yrs. Tuberculosis of the lumbar and low dorsal spine. Abscess. The child has been at the House of the Good Samaritan for two years. On entrance there was a large cold abscess which was opened. Tubercle bacilli were demonstrable in a smear from the pus and were grown on culture media. A few days after the abscess was opened he developed scarlet fever and was admitted to the Boston City Hospital. On his return he was treated a year in the wards with jackets. One year ago tuberculin treatment was commenced and as he had a secondary streptococcus infection of his sinus, he was given a streptococcus vaccine, made from his own organism, in addition to his tuberculin. His opsonic chart has been shown. There has been no local improvement of a permanent character. His sinus is still discharging and he has positive signs of phthisis and a suggestion of the development of an hypertrophic pulmonary osteoarthrophy.

CASE VI. Master C. 7 yrs. Tuberculosis of hip. Amyloid. Clinical diagnosis confirmed by histological examination after operation and by animal inoculation. In many ways this was a most remarkable case. The child came under observation at the Carney Hospital in less than a week after his first symptoms. Limp, pain, and muscular spasm, accompanied by a high temperature, led to a diagnosis of acute osteomyelitis. At operation a small amount of pus was obtained coming from a necrotic focus in the neck of the femur. The process extended both up and down the femur and a secondary operation was performed for the removal of more of the dead bone. This was succeeded by an acute nephritis which virtually became chronic. The spleen and liver were greatly enlarged and there was

profuse suppuration from sinuses in the thigh. The boy was soon in such poor condition that it seemed as though he might die at any time. Inoculation with 1–1000 mgr. of tuberculin was commenced and repeated on the average of once in ten days. Improvement, though slow, has been steady since the injections were begun. There is much less discharge from the abscess, his color is better, and his abdominal distention is greatly diminished. The urine also shows less of the products of a chronic nephritis than it did some months ago. This patient had a mixed infection with staphylococcus and was given a staphylococcus vaccine along with the tuberculin. About twenty inoculations were administered. There has been a decided improvement in the general condition of this patient, a change which it seems fair to attribute to the administration of tuberculin. This case would not have been so suggestive were it not for the fact that at the same time we had another child in the hospital in almost the same condition.

Case VII. F. S. This child is a boy of 5 1-2 years. He entered the House of the Good Samaritan in the fall of 1906. He had double tumor albus, both of which were suppurating. He was very pale and emaciated and had an enormous abdominal distention, part of which was caused by ascites and part by the size of his liver and spleen. There was a marked "caput medusæ." He had been having treatment under the best of conditions for the two years previous in the Children's, at Lowell and Bumkin's Islands, and failed to respond at all; in fact, had steadily grown worse. In desperation we commenced the administration of tuberculin, much against my better judgment, as it seemed as though I should be subjecting the child to an unwarrantable discomfort. He began to improve after a very few injections and under no different constitutional treatment than he had been having for at least two years. He is not now recognizable as the same child who entered the hospital seven months ago. He is fat, of good color; his sinuses are nearly healed; his abdomen is soft and very little enlarged; the area of liver dullness has diminished, and though both spleen and liver are palpable, they are greatly diminished in size. Both

knees are nearly completely ankylosed, but in very good position. He has had tuberculin administered with more or less regularity since his entrance. This case seems certainly to have benefited in a most extraordinary manner by the use of tuberculin.

CASE VIII. F. K. Six years. Tuberculosis of both hip joints. Abscess. Erysipelas. Child was under treatment at various places for a fairly active tuberculous process in both hips. The left one suppurated, and during the course of the treatment the sinus became infected with streptococci, and she had quite a virulent erysipelas. A culture from her own sinus gave us the opportunity to make a streptococcus vaccine from her own organism, and this was administered along with her tuberculin injections. In this case the pus burrowed out below Poupart's ligament on the anterior surface of the thigh, and though opened freely two or three times, under ether, refused to cease discharging. After the commencement of vaccine injections there was a very rapid diminution in the discharge until it finally ceased altogether. She was in the hospital about 5 1-2 months. Her chart has been shown. The progress of the tuberculous phase of her trouble was not affected by the use of tuberculin in any way.

CASE IX. A. McD. 4 1-2 years. Tumor Albus. Abscess. Flexion and subluxation deformity of the knees. This child was transferred from the Convalescent Home of the Children's, where he had been under treatment for a year or more. His general condition was excellent. The knee itself had six or eight sinuses, from which there was moderate discharge. The joint was very much swollen, caused chiefly by bony thickening. A culture from his sinuses gave a pure culture of the staphylococcus. From this a vaccine was prepared and administered along with tuberculin during the fall and winter of 1906-7. During the past three months he has been at the seashore hospital at Marblehead, and his sinuses are reported closed. During this time he has not been having tuberculin. I could not observe any unusual response to treatment in this case.

CASE X. Miss P. 12 years. Tuberculosis of left

hip. Abscess. This child entered the hospital with a very acute hip. She came from poor home surroundings and had had no adequate care. The thigh was flexed to a right angle with the body and traction would not overcome the spasm. Ether was given and the position corrected. Later a deep seated abscess appeared and was opened. From the time that I manipulated her in October, 1906, she commenced to fail. Tuberculin was administered before the manipulation and was continued through the fall and early winter without any effect; the patient became generally infected and died in March, 1907, of tuberculous meningitis. In this case I made the mistake of manipulating the hip joint and precipitating into the child's general circulation more tuberculous toxines than she could neutralize. I very much question in view of that fact if it was proper to have given tuberculin. This catastrophe should not be cited as an argument against the employment of tuberculin, except in cases of a similar character.

Case XI. Mr. B. 45 years. Tuberculosis of left hip. Operation. Clinical diagnosis was confirmed by histological examination. Patient was a longshoreman, who had a rapidly developing ankylosis of his left hip, without shortening. An X-ray showed nothing suggestive of tuberculosis and it was regarded as a non-tuberculous, infectious process. Under ether the hip was manipulated. The motion gained was not preserved. Suspicion of tuberculosis again being aroused, he was given a diagnostic dose of tuberculin and had a sharp constitutional reaction but no exaggeration of local symptoms. An open operation was performed upon the hip later, and a number of spurs removed from the superior lip of the acetabulum and a small abscess was exposed in the region of the gluteal bursa. The pathological report from this was tuberculosis. Injections of tuberculin were commenced and continued through the summer, fall and early winter of 1906–7. The patient gradually failed, developed signs of phthisis, and died in the early Spring of 1907. Again, in a case of this sort, the lesson it seems to me is that tuberculin is not to be administered, whatever other indications there may be upon estimates of the opsonic power of the patient's serum. Both this case and Case X were treated according to their opsonic indices.

In this series of eleven cases, therefore, we find four who were not benefited at all. Two were apparently most extraordinarily improved, and still another (Case III) experienced an improvement which, considering the gravity of the symptoms, may fairly be classed as attributable, in large measure, to his vaccines. Two have died and a third is dying. It is an open question if another (Case V) has not been made temporarily worse by the treatment. If we take out the two cases who have died, and it is fair to do this because they were cases in which the surgical measures employed may have imposed more of an infection upon the patients than it was reasonable to suppose any vaccination could take care of, there are only two cases left to speak positively in favor of the employment of tuberculin in the treatment of bone and joint disease. My experience, however, leads me to wish to give the method a longer trial, and with a more judicious selection of cases I believe better results may be forthcoming.

If the outcome of vaccine treatment has not been brilliant in tuberculous lesions it has been even less so in the case of non-tuberculous lesions.

In chronic polyarthritis it would be the greatest possible boon if the opsonic test could be employed to discriminate between types of infection. If this could be accomplished with any degree of certainty, the next step, that of making a bacterial vaccine for purposes of treatment, should be a comparatively easy one.

In the earlier part of our experience with opsonic work there seemed to be considerable to suggest the possibility of a solution of the problems in the diagnosis and treatment of chronic polyarthritis. When, however, one regards the cases through a long perspective, there is not much to give encouragement at the present time. In acute infectious lesions of joints, such as gonorrhœal arthritis, encouraging reports are coming from such reliable sources as the Johns

Hopkins Hospital and the Loomis Laboratory in New York, but the chronic problem seems to be a very different one.

Schüler, in Germany, has isolated from a very large proportion of the cases upon which he has been privileged to operate, a bacillus which he regards as the specific organism of chronic progressive polyarthritis. Three years ago Fayerweather isolated from the knee joint of a patient, upon whom I had operated at the Carney Hospital, an organism which he has described* and which Schüler† has declared from the description in Fayerweather's article to be identical with his baccillus.

The cultures of this organism had been preserved in the Tufts Pathological Laboratory, and we have been working with a vaccine which Dr. Leary has prepared from these cultures, in an attempt to diagnose and treat the type of polyarthritis with which, according to Schüler, this bacillus is constantly identified. All that can be said as a result of practically a year's experience with the application of the opsonic test for this bacillus is that we have been able to obtain a characteristic serum reaction in the blood of nearly all the patients who were possessed of the clinical signs of this type of polyarthritis. When it has come to treatment I think it is fair to say that there has been no considerable degree of improvement in any case, at least none which is not frequently seen in cases treated by other methods.

The fact that there has been such a uniformity in the opsonic findings in cases that are clinically of one type, together with this further observed fact, that when inoculated with a vaccine made from this organism they reacted locally, just as tubercular joints will react locally to too large an injection of tuberculin, it would seem that a lead had been given which it is worth while to follow.

*R. Fayerweather.—Am. Jour. Med. Sciences, December, 1905.
†Schuler.—Am. Jour. Med. Sciences. August, 1906.

The following cases are reported to show the kind of problems which have been attacked rather than to record favorable results of treatment.

CASE XII. Mrs. L. 40 years. Infectious polyarthritis. For five or six years the patient has been gradually becoming crippled in all of her principal joints and has been a great sufferer. With it all has come a considerable degree of neurasthenia. The joints are swollen, only slightly deformed, and possess very little motion. There is occasionally some redness over the phalangeal joints. Patient has been able to sit up in a chair, but walking has been impossible except with the assistance of two persons to support her. All the possible sources of infection were investigated to make sure that there was no present infective focus. A blood culture was made and found to be negative. Cultures from the stools were also made, but the investigation along this line was not completed. The opsonic index to the staphylococcus aureus, the streptococcus, and Schüler's bacillus of villous arthritis, was determined. The reports showed that the index was low, at various times, to each of these organisms, and vaccines were given for considerable periods, being graduated according to the response of the patient's serum to the inoculations. After approximately five months of such treatment there has been no appreciable improvement in the patient's local condition, which could be attributed to the vaccines. I think it is fair to assume, therefore, that the inoculation treatment in her case has been a failure.

CASE XIII. Mr. F. 48 years. Chronic, progressive, infectious polyarthritis. Three years duration. No present source of infection made out. Eighteen years ago had gonorrhœa. Nearly all the joints in the body have become involved. No redness or other signs of acute inflammation. Marked deformities of the knees and limitation of motion in all the joints which have been involved. A blood culture was sterile. The patient's index was low to Schüler's organism and also to the streptococcus. No improvement attended the administration of vaccines from either of these organisms. During the treatment the patient has developed an iritis which has pretty completely impaired the vision in one of his eyes.

CASE XIV. Mr. S. 21 years. Chronic infectious polyarthritis. After an exposure to dampness and cold one year before entrance to the Orthopædic Clinic of the Carney Hospital, the patient began to have swelling, stiffness, and pain in both knees. This was rapidly succeeded by similar conditions in the wrists, fingers and feet. His index was low to Schüler's organism, but neither local nor constitutional condition was benefited at all by a few inoculations with a vaccine made from this organism.

CASE XV. Mrs. S. 38. Chronic infectious polyarthritis. Patient has had a chronic suppurating ear for several years. Following the birth of a child five years before, coming under vaccine treatment, she began to have trouble in several joints. The knees have become so badly flexed that she cannot straighten them completely. Her index was low to the streptococcus. The injection of a vaccine made from three strains of streptococci produced no alteration in the local condition, except that the pain was worse and there was greater stiffness immediately after the injections. The discharge from the ear practically ceased, but this may have been in consequence of careful treatment at the hands of an aurist.

CASE XVI. Miss L. 35. Chronic progressive polyarthritis. The patient has been more or less constantly afflicted for five years with painful, stiff joints, particularly the knees, wrists, elbows and ankles. The symptoms were not very acute at the time she came under observation. She had had two very badly infected tonsils, and they were both removed; she had also had acute sinus inflammation. Her index was determined to be low to the streptococcus and also to Schüler's organism, and she was given faithfully for several months a vaccine made from a triple streptococcus culture. She also received a number of inoculations with a vaccine made from Schüler's organism. Neither of these produced any permanent effect. At times she, as well as several other patients, seemed to receive some temporary benefit from the inoculation.

CASE XVII. Mr. W. 45 years. Infectious polyarthritis. For four or five years he has been gradually becoming stiffened in the joints of his knees and hands. One

year ago he had an attack of pneumonia, during which the swelling disappeared entirely from his joints, and when he commenced to get about during convalescence, he felt that his arthritis had entirely disappeared. This immunity from former joint symptoms was only temporary, for within a few weeks all his old troubles had recurred, and since that ti methe knees had flexed to such a degree that walking was painful and difficult. The patient's index was taken for the staphylococcus, streptococcus, pneumococcus, colon bacillus, and Schüler's bacillus. His index was low to the pneumococcus and Schüler's bacillus. Vaccines from these two organisms were repeatedly given, guided by the opsonic index, and no material improvement occurred, which could not be attributed to local and constitutional treatment coincidently carried out.

Case XVIII. Mr. S. 52 years. Subacute phlegmon. Two years ago while in Egypt had a very acute phlegmon of the cheek, for which he was operated upon in Cairo, and had at the same time very large and often repeated inoculations of anti-streptococcic serum. Since then, in various parts of his body, never in any joint, but usually in close proximity to some large articulation, he has had more or less subacute phlegmons. One of these close to the elbow joint was incised and a pure culture of the staphylococcus aureus was obtained. From this culture a vaccine was made and the patient was given several inoculations of this vaccine without any appreciable benefit.

The next two patients had intermittent hydrops.

Case XIX. Mr. R. 36 years. Intermittent hydrops. Psoriasis. For seven years has had at irregular intervals an intermittent swelling in both knees and both ankles. Associated with this condition has been a very general psoriasis. When, as has happened on several occasions, he has had freedom from joint symptoms for six months at a time, he has also been entirely free from psoriasis. The knees have caused him the greatest inconvenience. The right one has been the more swollen and has had a five day interval between attacks. The cycle for the ankles was not as clearly defined and he had never taken the trouble to determine it. The capsular swelling in the knees wholly

disappeared. The right knee joint was subjected to an arthrotomy with the hope that the cycle might be broken up. A culture taken from the interior of the joint gave the staphylococcus, which seemed a peculiar bacteriologic finding, but a vaccine was made from the culture and was regularly administered. For three months the operated knee showed no signs of an intermittent swelling. The left knee during this period was less inclined to swell, but did so to some extent. At the end of this interval the right (operated) knee returned to its cyclic distention, though the enlargement has not been as great as before. Observations upon the opsonic index at first showed that it was low to the staphylococcus, but later on the streptococcus seemed to be the organism which showed a low index, and accordingly a streptococcus vaccine was substituted, but without any alteration in progress of the trouble.

Case XX. Miss S. 30 years. Intermittent hydrops. Both knees have swollen alternately every seven days for over two years. Two days were required for the swelling to attain its maximum and recede. The swelling caused a little pain for a few hours, but chiefly inconvenience from the enlargement. Her serum was tested and found to be low to the streptococcus. A vaccine was injected regularly for some time, but not carefully watched with opsonic tests. The patient has been taking Fowler's solution alternating with quinine during this entire period. There have been two intervals of two weeks each when there was no appreciable swelling in either joint, but this has occurred before under other treatment and no treatment. The amount of swelling is decidedly less, though except for the two intervals cited there has been no intermission of the regular cycle.

Discouraging as these cases are, looked at from the clinical point of view, it must be remembered that we have been applying a new method, the shortcomings of which are not at present wholly understood. Circumstances have sometimes existed making it impossible that some of the conditions imposed by laboratory investigators could be carried out. It may be that cases have been treated with vaccines

made from organisms having no possible kinship to the organisms originally present in the patient, though the opsonic findings have pointed that way. Again it may be possible that some of the conditions treated were not originally caused by an infective agent. These and many other obstacles stand in the way of drawing conclusions from the results of the first year's clinical experience. A good many more cases were treated and tested than have been recorded here, but those selected for report were chosen because they covered the types of cases most satisfactorily. It would have been possible to select a group, perhaps an equally large one, where the conformity of the opsonic and clinical findings would have been entirely favorable to this new method of study. So in the matter of treatment, typical opsonic charts might have been exhibited corresponding to remarkable degrees of clinical improvement. The writer believes the danger of expecting too much from an untried method is greater than that of expecting too little, and is desirous of encouraging the continuance of trials now going on in the laboratory provided they can be checked by unbiased clinical observation.

DISCUSSION.

Dr. L. S. MEDALIA, of Boston: I wish to say that some of the cases that Dr. Painter has mentioned have not been under treatment since the first of February. There are a few cases at the Good Samaritan which have been treated up to date, but those at the Carney Hospital have not been followed up since February first.

I would emphasize the fact that this treatment has to be carefully and energetically carried out, for the conclusions drawn from those not followed up properly have been unsatisfactory.

ARTICLE XXXII.

THE PROSTATE FROM THE SURGICAL STANDPOINT.

By CHARLES S. BENSON, M.D.
OF HAVERHILL.

READ JUNE 11, 1907.

THE PROSTATE GLAND FROM THE SURGICAL STANDPOINT.

THE literature on the Prostate Gland is most voluminous. One can find carefully prepared statistical reports; papers proving to the writers' satisfaction the only and best method of operative attack. Others show conclusively that catheter life is the only way to handle these sufferers, until the outraged bladder settles the question in its own way.

For all exhaustive treatises, anatomy, functional physiology, histology and pathology, I refer you to the interesting works of Denver, Young, Willy Meyer, Guiteras, Murphy, Chetwood, Keyes, Watson, and a host of others. It is a simple matter to abstract any and all of these writers, but of what avail to you and me, for such would be inferior to the originals which lie within your reach.

My personal operative experience is confined to but few cases, all done by way of the perineal route, and all cured, which fact is of little value considering the small number, for the next few may go the other way.

As an interne, however, I assisted at no small number of prostatectomies, and cared for these afterward. Most were done supra-pubically, and I have since been rather prejudiced against this method, for convalescence was stormy and mortality was pyrotechnical, as I look back on the fistulæ, and infections of the pre-vesical space.

Since this paper was presented in the winter I have added two cases only, one of which is now two weeks in the hos-

pital and apparently doing well. This is my only case operated upon by the supra-pubic route, and for the reason that it seemed best to divide the operation into two stages.*

With only these few prostatectomies to my credit, it may seem presumptuous to address you, but I crave your indulgence for so doing and further ask your tolerance in that I may make these remarks personal, not abstractive, nor quotative; merely as the subject appears to myself. Suppose, then, we discuss the prostate gland along the following lines:

1st. Is the patient with a prostate which is symptomatic, better for being subjected to operation, or should he be advised catheter life as long as possible, followed by ultimate operation when catheter life is no longer possible or endurable?

2d. If operation is the desideratum, what then constitute the indications for operation?

3d. If not operative, is there any other treatment apart from or combined with catheterization?

4th. If operation, what shall be the method of attack?

5th, and perhaps this would have better come first, although I question it:—what constitutes the symptomatology of hypertrophy of the prostate gland which establishes the diagnosis?

6th. Prostatism without hypertrophy of the gland, its causes and treatment; the confusion of this with true hypertrophy of the prostate.

Concerning the first proposition, namely, catheter life or operation, there has been during the past several years a very great diversity of opinion as to the preferable means of procedure, and to-day it is still a very much mooted question, although I think surgeons are coming nearer together as to the advisability of the earlier operation.

* This case died of bronchitis some weeks later, supra-pubic wound not entirely closed, but clean and granulating.

For my own part, it seems to me after diagnosis is established, that the earlier we operate the better the chance of life, the better the chance of complete symptomatic recovery.

To pursue the old course of using the catheter as long as the patient could be made comfortable by such means, and ultimately operating when the patient's condition had become desperate from cystitis and ascending pyelo-nephritis, is wantonly running up the mortality rate and giving us simply palliative surgery. The fact that in individual work the mortality of all methods of operation is between four and six per cent. would seem to me a great argument in favor of the earlier operation, before we have infective complications.

The catheter is of the greatest use in establishing the diagnosis, and every patient should be kept under observation for a few months before deciding upon operative measures; but once the diagnosis is unqualifiedly established, the sooner we operate the better.

When I say diagnosis, I mean more than merely that a patient has hypertrophy of the prostate; I mean that this patient has more than merely nocturnal frequency of micturition. I mean more especially, that urination is difficult to start, is what we call stuttering and stammering during the act; stops slowly with considerable dribbling and soiling of the clothing, and that the relief which ordinarily comes from emptying of the bladder is not experienced by the sufferer from prostatic hypertrophy.

In other words, to go only a step further, we now use the catheter and establish that the patient has four or more ounces of residual urine after urination.

Now it is perfectly possible for this patient to have this amount of residual urine and not have cystitis. I believe that with these symptoms it is not necessary to subject this patient to years of catheter life in the future, but by oper-

ating at this time we can save this inconvenience and also get a symptomatic and actual cure.

In this connection it is only fair to say that there are certain types of cases in which operation is probably the last resort after the case has been considered most deliberately, and yet for the most part I think operation should not be undertaken or considered at all.

I refer especially to those cases coming to the practitioner, who have been so long sufferers from an enlarged prostate with its damming back of the urine, whether or not depending at the present time for their visit completely on catheterization. These patients have an atonic bladder, and operation will not cure or relieve symptomatically this condition. It should be undertaken only when there is actual danger of death, in patients comparatively young and to whom every year of life is of the greatest importance, knowing that although you remove the hypertrophy, you still go on to the end with the catheter, and your operation has been done simply to provide the means of introducing the catheter.

Another class where it seems to me operation is questionable, is in old cases which at the time of being seen, already possess infected bladder and kidney. By all means, in these cases, I should advise a long treatment by catheter and irrigation, unless the case is so desperate that suprapubic drainage affords quickest relief for the infected condition, if there is any relief at all to be obtained. These last cases can only be judged on their individual merits.

There is no dogmatic position of No operation; or, Operation. One thing is very certain, however. Any operation which takes more than a few minutes to perform, will certainly put your patient on the wrong side of the mortality statistics.

To take up the next topic: The desirability of operation being established, what then are the indications?

It almost seems as though I had answered this question in the preceding paragraphs, but it will do no harm to repeat.

For me, at least, I believe that operation is indicated, when, upon rectal examination we find a pronounced enlargement of the prostate gland.

When there is difficulty in starting the urinary stream, and I might say, difficulty in stopping the same, with or without necessity of using the catheter. In addition to this, the discovery of a few ounces of residual urine. These I think, after proper observation, justify advising the patient to submit to operation and enable the surgeon to hold out the prospect of a cure and to inform the patient, that taken at this time the risk is very much lessened.

To take in topic three and six under one head, namely: If the case is not operative, is there any treatment apart or combined with catheterization and the subject of prostatism without hypertrophy.

It is impossible to separate these two for the very reason that the only justification of adopting a treatment other than catheter life or operation in hypertrophy of the prostate gland, is the fact that hypertrophy does not exist; that we do have a condition which I have diagnosed as prostatitis, in which there is a certain amount of irritation and congestion of the gland, without any increase in glandular and connective tissue elements, which in itself gives rise to very similar symptoms of the early stages of hypertrophy. Perhaps if anything, this condition carries with it more pronounced discomfort than the graver type.

Given then, prostatism of this nature, what are we to do with it?

If we consider the fact that in the majority of instances this is due to an old gonorrhea, we shall come nearer appreciating the treatment which will be most efficacious. We will massage the prostate gland and use instillations of nitrate of silver, deposited in the membranous and pros-

tate urethra. We will use, I say, instillations, not injections, and I wish you to consider this word instillation as spelled with capital letters. It is only a few drops that should be thrown in, and the strength of the solution should be varied according to the indications arising during the course of treatment.

It is rarely that irrigation of the bladder itself will be called for. I might say it is never called for, except as we may irrigate the whole urethral tract; in the milder type of cases, filling up the bladder only that we may get a second passing of the fluid over the area selected when the bladder is emptied.

Perhaps in this connection it will be well to touch upon the possibility of treatment in cases followed with cystitis, ureteritis and pyelitis.

Kelly, of Baltimore, in examination at one time practised washing out the ureters and the pelvis of the kidney through catheters passed clear to the renal pelvis. A few other surgeons have tried this method of procedure, but for the most part, however, have condemned it.

I cannot help believing that it is extremely dangerous, even in the most skilful hands, and certainly it is entirely out of the province of the average practitioner who neither possesses the equipment nor the training to even catheterize the ureters; consequently in these severe cases we are practically limited in our treatment to vesical drainage alone.

4th. Now if we have decided upon operation, how shall we proceed?

We have practically only three operative procedures before us, namely: Bottini, suprapubic, and the perineal. I purposely leave out what is known as the combined method, as being absolutely useless and dangerous in creating an unnecessary opening.

The Bottini operation has, I think, a place, but the limi-

tations are very strict, and it requires a high amount of technical skill and training. It is adapted only to a very mild degree of hypertrophy, or perhaps I might better say to those cases which do not necessarily possess a true hypertrophy of the prostate, but only a stenosis of the vesical orifice, or what is known as the vesical bar. Its great advantages lie in the fact that generally anæsthesia is not needed, that the patients are not confined to bed, or even, but for a short time, to the house, yet it is dangerous. It necessitates, also, conditions which allow of a painstaking and minute cystoscopic examination. For the most part this operation has been again assigned to oblivion and will, I trust, remain there. With very few exceptions, it possessed no merits which are not equalled or surpassed by prostatectomy; its only virtue apparently consisting in adaptability to a very small number of cases wherein general anæsthesia is absolutely contra-indicated, and any measure which may give relief is justifiable, short of electing actual operative death.

Certainly the suprapubic operation has its uses.

Personally, I think that in those cases of badly infected bladders, in which the vital tone is very much lowered, that a suprapubic opening done at first only for drainage, and irrigation of the bladder, to be followed after an interval by an enucleation of the prostate gland through the same opening, is the only proper method of procedure in these cases.

We are told that the large, soft prostates are better removed through a suprapubic opening; that the small fibroid prostates are better removed through the perineal opening. We are told by the advocates of suprapubic operation, that the urethra in part is better preserved by suprapubic operation. Yet on the other hand, within two years that party who popularized the suprapubic operation is now pleading for removal of the prostate gland en masse,

together with the prostatic urethra, of course thus dropping one of his own arguments for the superiority of the suprapubic operation.

We are told that the operation is quicker and the drainage better by this route. I question very much if the operation is any quicker in uncomplicated cases. I question if drainage is not better or as good where the operation and the drainage are sought for at one and the same time in those cases which permit of doing the operation in one stage; certainly not where we are forced to operate in two stages.

For the perineal route which has been so strongly advocated by Young, it is said that we can see the parts as in any dissection. The same claim is made as for the suprapubic route, that the drainage is better by the perineum. It is further claimed for the perineal operation that we can preserve ejaculatory ducts, and so preserve the potency of the patient. Now as a matter of fact, I reserve for myself the same right in the election of the method of operation as I would reserve for myself if I were practising medicine. If I were an internist and I knew of any drug which would benefit my patients, whether used by the homeopathist, or the eclectic, or the so-called "herb doctor," I should use it; in operating on the prostate gland, if it seems best to me to operate by the suprapubic route, I shall do so. If I elect the perineal it will be because I think that I can give the patient better prospects for an ultimate cure with less risk to life. I do not believe that it is necessary to make a minute and pains-taking dissection of the perineum, nor attempt in the vast majority of cases to preserve the ejaculatory ducts.

Most of our cases are in men well past sixty, and at the time that they come to us these patients are impotent, not only as regards their powers of procreation, but as regards their power of sexual relation, and the conservation of these powers is a matter of absolute indifference.

The median perineal incision, with opening of the membranous urethra, dilatation of the vesical neck and enucleation of the prostate masses through the lateral walls of the urethra can be done rapidly and thoroughly and will give one as perfect results as any other method of operation. There is rarely any hemorrhage, if we keep with the capsule, using the finger as a blunt dissector.

There are certain cases in which the relation between the capsule and the gland and the surrounding tissues is so intimate that enucleation as such cannot be accomplished. It is said that these cases can be better removed by morcellement than through a suprapubic opening.

This means rather a wide destruction of the vesical mucosa.

For my part, I think that these masses can be removed through the perineal incision, modified to give a broader exposure of the field, and it is in these cases that I believe in the technique of Young, using a tractor within the bladder, as causing less destruction of the bladder tissue than can be accomplished in any other way. It seems to me that the question of injury to the rectum has been very much magnified. If we use the exaggerated Trendelenberg position, with the thighs strongly flexed on the abdomen, we shall find that the rectum falls away very readily from the prostate. The only question of anatomy that I shall raise in any operation through the perineum will be to keep close to the bulb dividing the recto-urethralis muscle and keeping outside the fasia of Denonvillier.

One thing which has run through the entire list of topics, if you notice, very much like a motif in music, although not named, has been the question of diagnosis. Beyond what has already been said of this, I am disposed to say a little something as regards the use of the cystoscope and also touch upon the differential diagnosis.

There is one condition in which the cystoscope is of very

great value; that is where we have an enlarged middle lobe, with comparatively small amount of hypertrophy to be felt by rectal touch.

If we can get the instrument into the bladder at all, it gives us a very good view of the extent of this enlargement and enables us to choose our method of attack with reference to the ease of reaching the trouble. On the other hand, to use the cystoscope needs a large amount of special training, or the observations are not only not of value, but they are absolutely dangerous. These cystoscopes which depend upon air inflation of the bladder, and in which the opening or the prism is situated at the end of the tube, the so-called direct cystoscope, is of no use in this condition. We need a simple instrument with a prism on the concave side and water dilatation of the bladder. To use the instrument with any satisfaction, we must follow the procedure of Young, of Johns Hopkins, who has devised a scheme for reconstructing the inverted cystoscopic image and patching together the detached picture segments to give us a schematic representation of the whole field.

In the average case of prostatic hypertrophy the cystoscope is of no practical value. There is, however, one condition of the bladder as determined by the cystoscope which justifies us in diagnosing prostatic hypertrophy without being able to feel same. I refer to the trabeculated bladder, which almost invariably is significant of enlarged prostate, with more or less urinary retention.

It is particularly important to differentiate hypertrophy of the prostate gland from new growths of the same, especially malignant disease; although in cancer we are told that hematuria is one of the symptoms, yet it occurs perhaps in not over twenty-five per cent. of the cases.

In the early stages the most prominent symptom for differentiation is pain. The pain of cancer is much more severe that the pain of advanced hypertrophy, if such occurs;

is sharper, more lancinating and very apt to radiate in the groin and down the leg. In fact this radiation, if we omit the coincidence of stone, is almost pathognomonic of cancer of prostate. A little later we get the stony hardness so significant of malignant disease. My records give me a total of eleven cases of malignant disease of the prostate, only one of which, so far as they have been followed, has shown hematuria. All but two have had severe pain; in fact that was the first symptom noticed by the patient, and for which he sought relief. The increase in size of the prostate had not been very marked prior to the onset of cachectic symptoms.

Manifestly it is important to exclude malignant disease before we decide upon operative measures, for with the one exception where the cancer is contained entirely within the capsule of the gland, operation is absolutely hopeless.

Then, too, simple hypertrophy of the prostate gland, or a congestive condition which has passed beyond the actual acute stage, calling as they do for two entirely different lines of treatment, should be carefully distinguished.

Retention of urine, difficulties in urination, in fact practically all the symptoms that go with hypertrophy, are present in this congestive state. Even residual urine can be found, without the evidence, however, of any cystitis. This condition may persist for many weeks. Its treatment, however, is merely the employment of massage, instillations to the membranous and prostatic urethra; perhaps also enemata of hot water or the irrigation through a metal closed tube of hot water by way of the rectum.

Then, too, we should differentiate between a primary pyelo-nephritis, to which the cystitis is secondary in a man well past fifty, who in the natural order of events would possess a prostate gland which to the touch is more or less enlarged. Exclusive of the actual retention of urine, he may possess all the symptoms of the sufferer from prostatic

hypertrophy, and only the most careful consideration of the previous history will help to establish the diagnosis, and it is in this type of case where operation for the prostatic trouble is practically almost always contra-indicated.

We cannot use a cystoscope to catheterize the urethra, on account of the dangers of increasing the infection and causing uræmia; nor is it going to be of very great value should we succeed in securing the urine from one or both kidneys, for the reason that the whole question hinges on the fact of the infection being of an ascending or descending character. As regards the existence of stone in the bladder, the operation for relief of the hypertrophy of the gland will serve for the removal of the stone. In other words, there is a limit to the size of the stone which may be removed by way of the perineum. There is practically no limit to the size of the stone which may be removed by the suprapubic operation.

Abscess of the prostate gland may simulate acute prostatitis, and with the exception of tuberculosis and what is practically a chronic abscess, comes into conflict with the prostatic hypertrophy but very rarely. Save as in massive acute abscesses, I think that the ordinary prostatic abscess should be evacuated through a perineal incision and not by the old method of a curved trocar thrust into the abscess through the rectal wall or by incision through the rectal wall. Incision through the rectum is, however, I think justified in those large abscesses which pronouncedly bulge into the rectum and often by pressure bring the rectal walls practically together.

Cysts of the gland require no especial mention. If large enough to give symptoms, they require evacuation and, of course, are better done through perineal incision. It is largely a matter of indifference whether we diagnose cyst of the prostate or hypertrophy of the prostate.

If we find a cyst we stop with our evacuation. If we find hypertrophy we simply proceed and enucleate the gland.

Tuberculosis of the prostate is rarely a primary disease, usually being secondary to tuberculosis of the testicle and seminal vesicles, and in those rare instances where we find it as a primary focus, its symptomatology will be largely that of abscess, either acute or chronic, and its treatment will be surgical only in so far as it is an abscess.

During the year, in the course of my office practice, I see a great many cases of chronic gonorrhea with all its complications, and I find that daily I am either examining or treating by massage several cases in which there is apparent enlargement of the prostate gland.

These cases, of course, are simple cases of prostatitis, and yet they are undoubtedly on the road which will bring them in later life to hypertrophy of the prostate. Coincident with these cases, and really the cause which keeps alive the prostatitis and the gonorrhea, is inflammation of the seminal vesicles, and it is really by the emptying of these vesicles, through massage, that we improve and cure the gonorrhea and to a large extent the prostatitis, although in long-standing cases it is my opinion that changes have taken place in the prostate from which the gland never recovers.

An analysis then of what I have said to you to-day resolves itself practically into the following: A plea for the education of the public as we meet members of it in our offices, in regard to the dangers of neglected prostatic hypertrophy. A plea for the earlier recognition by the physician and surgeon, and after a careful hearing of all the evidence, the advice to the patient of a much earlier operative interference than has heretofore been the custom, for the sake of cure of symptoms and conditions and the danger to life itself. A plea not to send the patient to a surgeon who elects, or to perform yourself, a suprapubic or a perineal operation, without due regard for all the facts in the case, but to advise your patient and so do yourself that which shall be the

best for the individual concerned, remembering that time in operative work is a very great feature, in the fact that it lessens anæsthetic shock in a patient already on the downward path of life.

Of course it goes without comment that this saving of time shall not be at the expense of clean, smooth work, nor is it necessary on the other hand to exaggerate infinitesimal details.

There is no place in the surgery of to-day for either the putterer and bungler, or the man who is all "front" and gallery play, when dealing as we are with Life, attempting to the best of knowledge and ability to keep it from flutter- into the "Great Beyond."

ARTICLE XXXIII.

TWENTY-FIVE YEARS' EXPERIENCE IN THE TREATMENT OF STRICTURE OF THE URETHRA.

By FRANCIS S. WATSON, M.D.
OF BOSTON.

READ JUNE 11, 1907.

TWENTY-FIVE YEARS' EXPERIENCE IN TREATMENT OF STRICTURE OF THE URETHRA.

WHAT I have to say upon this subject to-day is of a frankly personal nature, and relates in large measure to my own experience in the treatment of this malady and some of the lessons that I have learned in the course of the last twenty-five years during which such cases have come under my care. For the sake of clearness and to enable you to follow my remarks more readily, I will state the points upon which I shall touch and the sequence in which they will be referred to, which is as follows:

1. The cases summarized in the form of a table which shows their total number, the nature of the process in which the strictures originated, the different methods of treatment to which they were submitted, and the mortality attending each of them respectively, and of the total number as a whole.

2. An analysis of these cases with reference to the causes of death.

3. The character of the results of each of the different kinds of treatment.

4. The rules which must be observed in the performance of internal urethrotomy and the after care of the operation.

5. The best manner of dealing with impermeable or very dense and extensive strictures of the perineal portion of the canal.

TABLE I.

	Cases.
Of gonorrhœal origin	542
Traumatic	35
Due to the action of caustic	1
Congenital	2
	580

Methods of Treatment.

	Cases.	Deaths.	Mortality.
Gradual dilatation	172	1	0.58%
Divulsion	9	0	0.00%
Internal urethrotomy alone	220	2	0.91%
Internal and external urethrotomy	102	5	4.9 %
External perineal urethrotomy alone	56	5	8.9 %
Combined suprapubic cystotomy and external perineal urethrotomy	4	0	0.00%
Partial resection	4	0	0 00%
Electrolysis	13	0	0.00%
Total	580	13	2.2 %

Operative Mortality.

Gradual dilatation is universally acknowledged to be the safest of all methods of treatment.

The mortality attending the operations of divulsion and internal urethrotomy is very small and is practically the same for both of the methods.

The high mortality which attends the performance of external perineal urethrotomy is to be referred to the critical conditions under which the operation is in many cases performed, and not to the procedure *per se*.

Electrolysis has about the same risk attaching to it as that which attends gradual dilatation.

The mortality of divulsion and of internal urethrotomy I have determined from a series of 1000 cases of the former and nearly 5000 of the latter, which I have collected from the literature.

The operative mortality of the series of cases in which divulsion was done was 2.1%. That shown in cases in which internal urethrotomy was done was 1.9%.

In my own series of 220 operations of internal urethrotomy in which that operation was performed alone, there were but two deaths, or less than 1.0% mortality, and I have never had a death follow the operation in private practice.

The mortality of external perineal urethrotomy I have computed from a series of 2200 cases which I have collected from the literature and found it to be exactly 9.0%. My own mortality record is almost identical with that of this large number of cases, it being 8.9% in 56 cases in which I have done the operation alone.

Causes of Death.

Deaths occurring in connection with any of the methods of treatment of stricture are, practically speaking, referable to one of two conditions.

1. Shock propagated from the peripheral nerves of the urethra and originating in the use of urethral instruments. One of the interesting features in this connection is the curious fact, which is true of both the constitutional disturbances and of the deaths which follow the use of urethral instruments, as in gradual dilatation for example, and which is, that neither the one nor the other of these untoward results is more likely to occur from shock, when violence and injury have occurred with the use of the instruments, than when they have been used with skill and gentleness.

In the cases in which deaths of this kind take place, the renal function is almost always suppressed. In most instances it is probable that the kidneys are already more or less diseased, but it is still an open question whether or no nervous impulses transmitted from the urethra or bladder in this way are capable of suppressing the function of a normal kidney.

The deaths of the kind of which I have just been speaking occur usually within a few hours following the use of urethral instruments.

2. Sepsis in one form or another accounts for almost all the other deaths which occur. In the cases in which so-called urinary infiltration is present, it is the already existing general septicemia which is responsible for the fatal result, and not the operation.

Much the larger number of deaths occur in this class of cases.

Death from sepsis is undoubtedly more likely to follow internal urethrotomy than other forms of treatment, if cleanliness and aseptic precautions are not observed in the performance of the operation. If they are, this ceases to be true and no further proof of the fact is needed than the very small mortality which appears in connection with the large number of operations which I have mentioned above.

As a matter of fact and every day experience, a large contingent of the deaths is furnished by the employment of gradual dilatation by those who are unskilled in its use. This is because of the making of false passages by the rough use of the instruments and the opportunity thus afforded for the originating of septic processes which have their starting point in the false passages and the septic products which they harbor.

Gradual dilatation is therefore quite as dangerous a method of treatment as any other when it is not carried out by well trained hands. But few practitioners hesitate to use this method while they would not undertake the more definitely operative measures.

Character of the Results Obtained by the Different Methods.

At the time at which I was a medical student one often heard the dictum, "Once a stricture, always a stricture;" in other words, it was regarded as an incurable malady. How little true this is to-day we shall see in a moment.

Cure.

By cure I mean that patients do not require to pass

sounds subsequent to the operation or to the completion of the treatment, in order to maintain the normal calibre of the urethra.

My knowledge of the later results obtained by *gradual dilatation* by *internal* and by *external urethrotomy* is derived from my own experience, and that is the most accurate basis upon which to rest an opinion. Of *divulsion*, my own experience is not sufficient to enable me to speak authoritatively, I have therefore taken for the purpose of determining the final results of this method, the most favorable exhibition of them with which I am familiar, namely, the table of twenty-seven cases out of a series of 404 operations performed at the Massachusetts General Hospital and published a number of years ago by Scudder. Before referring to these two methods, I will dispose in a few words of the results obtained by *gradual dilatation*, and *external urethrotomy* and *electrolysis*.

I have succeeded in curing but ten out of 172 patients treated by gradual dilatation, that is to say just a little less than 6.0%, and in all ten of these cases the strictures were in an early stage of formation.

Most of the 56 patients upon whom I have performed external urethrotomy alone were hospital patients and the larger number of them were lost sight of. Of the few that I have seen and examined since, but a small proportion have been cured, 10.0% perhaps.

When electrolysis was being vaunted as a method of treatment, a number of the members of the American Association of Genito-Urinary Surgeons gave it a fair and thorough trial. Personally I applied it in 13 cases. The unanimous verdict of all of us was that it was useless in all cases and harmful in some.

Internal Urethrotomy.

Of the whole number of 322 patients upon whom I performed the operation of internal urethrotomy alone, or in

combination with external urethrotomy, there were 96 who furnished the opportunity to make subsequent examinations of the conditions of the urethra. In 22 of these cases less than two years had elapsed when these examinations were made since the operations were done, and I have not included them in the table. In 20 *others* the operation *failed*, in that the symptoms were not wholly relieved, or else that recontraction had taken place. In the remaining 54 cases, or 56.0% of the whole number, *cure* had resulted from the operations. The subjoined table gives in detail

Cases.	Calibre of strictures when operated upon.	Times at which later examinations were made.	Sizes to which strictures were cut.	Calibres when examined later.
2	22 and 24.	22 Years.	32 and 34.	30–30.
2	26 and 14.	20 "	32 in both.	30–29.
3	25–27 and 22.	16 "	30–32–32.	30–28–28.
2	20 and 18.	15 "	30 and 32.	30–29.
1	18.	13 "	34.	30.
3	12–10 and 26.	12 "	32 in all.	32–30–28.
2	22 and 18.	10 "	30 and 32.	28–30.
2	18 and 24.	9 "	32 in both.	32–30.
5	14–18–20–10–22.	7 "	32 in all.	32 in 1; 30 in 2; 29 in 2.
5	8–26–24–14–18.	6 "	32 in all.	30 in 3; 28 in 2.
8	Fili–22–14–22–18 22–16–10.	5 "	34 in 2; 32 in 4; 30 in 2.	32 in 3; 30 in 2; 29 in 1; 28 in 2.
8	22–20–26–24–20–10. Fili–26.	4 "	32 in all.	30 in 6; 32 in 1; 28 in 1.
11	28–14–22–10–16–	3 "	30 in 2; 32	30 in 4; 32 in 3;
—	Fili–22–14–24–		in 9.	28 in 4.
54	22–26.			

No contraction whatever in - - 13
Recontraction of 4 mm. in calibre in 12
" " 3 " " " " 5
" 2 " " " 20
" 1 " " " " 4—54

the calibres of the strictures at the time of operation, the times at which the examinations were made subsequent to the operations, the sizes to which the strictures were cut by the operation, and the calibres of the urethras throughout when examined later.

It is to be understood that sounds were not used in any of these cases subsequent to the operations except at intervals of a year or more for the purpose of examination.

The following are the data given in Scudder's table:

Cases.	Times elapsed since operation.	Use of Sounds.	Recontraction or not.
2	12 Years.	Yes.	28 in both.
1	11 "	No.	16
1	9 "	Yes.	21
1	8 "	Once in 6 months.	22
1	7 "	Yes.	29
1	6 "	"	30
2	5 "	In one case, 4 months; in the other, no.	30 26
6	4 "	In 1 case, no; in the other 5, yes.	13 25–21–25–18–31.
3	3 "	In 1 case, no; in 2 others, yes.	Very small in one; in other 2, 30–21.
3	2 "	In one case, 2 months; in 2 others, yes.	26 in 1; in other 2, 26–11.
6	1 "	In 4, yes; in other 2, 6 mos. and 9 mos.	28–30–25–25 in 4; in other 2, 26–29.
27			

It will be seen from this table that there were but four patients who did not use sounds at all after the operations. Recontraction took place in all of the cases as follows:

To 26, 16, 13, and to a "very small size." That is to say, none of them can be called cured.

In five more of the cases the patients used sounds but a short time only, or at long intervals after the operations. In three of these cases no recontraction of any consequence

took place, and they may properly be claimed as cures. In the other two cases recontraction to 26 and to 22 occurred.

The rest of the patients, 18 in number, all used sounds after the operations. Even so there were but eight of them who succeeded in maintaining the urethral calibres at sizes varying from 26 to 30. Recontraction in various degrees (between 11 and 25) occurred in the other 10 cases despite the use of the sounds.

In other words 11.0% of cures by divulsion as compared with 56.0% in internal urethrotomy.

With the exception of a very few English surgeons and a small number of the profession in this community who still practice, I believe, the operation of divulsion, it has been for many years universally relegated to the number of obsolete surgical procedures to which it properly belongs.

Why should the results of internal urethrotomy be better than those obtained by other forms of treatment, and why should not the cicatrix of an internal urethrotomy incision follow the law of other cicatrices and contract?

These questions have been renewed from time to time ever since Reybard, in 1853, presented in his treatise upon urethral stricture the results of his study of the cicatrization of *longitudinal* incisions in the urethra.

He showed conclusively that such wounds, if prevented from reuniting during the process of healing, result in smooth, thin, delicate, and non-contractile cicatrices which add to the calibre of the urethra at the points occupied by them, instead of narrowing it.

The truth of this statement was confirmed for the first time by post mortem examination, in the human subject by Perrin in 1865, in the case of a patient dying three years after an operation of internal urethrotomy performed upon a stricture of the bulbous portion of the canal.

Since then other post mortem proofs have accumulated,

and a very large number of observations in the living subject by means of the endoscope and other instruments have established beyond dispute the correctness of Reybard's assertion.

Not long ago I had the opportunity of examining two patients upon whom I had performed internal urethrotomy 22 years previously. Both had had well marked organic strictures. Neither of them had used sounds subsequent to the operations. The endoscope enabled me in one case to detect a very fine white line upon the roof of the urethra at the site of my former incision. In the other urethra I could find no evidence of any cut having been made. There was no sign of stricture in either case. Acorn tipped sounds failed to detect the slightest unevenness or constriction in either of them. In both cases a number 30 sound slipped perfectly easily into the bladder through the urethra.

It is evident why internal urethrotomy is the method of choice from a surgical point of view. Let me add, however, that this is true only with certain reservations, and only when the operation is done according to certain rules which will be specified in a moment.

Gradual dilatation remains, as has been said, the safest of the procedures. There are three reasons for it not being the form of treatment which is always applicable. These are:—

1. It is rarely curative. 2. It cannot be applied or it cannot be made effective in cases of impassible stricture, in such as are so resistent as not to yield to the pressure of the instruments introduced, and in such as recontract rapidly after the employment of the instruments used to dilate them—resilient strictures. 3. It is unwise to employ the treatment of dilatation when constitutional disturbances, chills, fever, etc., follow its use.

The cases which fall under one or another of these headings are numerous and all of them should be treated by in-

ternal urethrotomy. Upon one point I wish to lay special stress. It was insisted upon most wisely by Sir Henry Thompson, and is this. In the cases in which gradual dilatation is attended by constitutional disturbances, it is far safer to perform an internal or an external perineal urethrotomy than to continue the treatment by gradual dilatation.

I accentuate this because I know it to be true from experience, and because those who may be ignorant of the fact might well hesitate to adopt the seemingly more radical procedure of internal urethrotomy in the face of the occurrence of constitutional disturbance which had arisen in the course of gradual dilatation.

On the other hand the following restrictions should be imposed upon the operation of internal urethrotomy.

1. It should not be applied to strictures further from the meatus than five inches. This is because of the greater danger of hemorrhage and of sepsis which occur when the incisions are made at a deeper point than this.

If external perineal urethrotomy is *combined* with internal urethrotomy, the latter operation can be performed as safely upon the strictures of the deep urethra as upon those of the anterior.

2. The operation is not as effective as external urethrotomy or resection in cases of very dense and extensive strictures of the perineal part of the canal and deeper.

Rules to be Observed in the Performance of Internal Urethrotomy.

To be safe, and if it is to be successful in obtaining cure, the following rules must be strictly observed in the performance of internal urethrotomy.

1. The urethra must be thoroughly irrigated immediately after the operation, after the first urination and after the first passage of sounds subsequent to the operation. The bladder should also be emptied and irrigated at the end of the operation.

2. The meatus must be cut to a size of 4 mm. larger than the normal calibre of the urethra.

3. The strictured part of the canal must be cut to the full size of the normal calibre of the urethra in each individual case.

4. The normal calibre should in each case be determined beforehand in accordance with the standard of measurements laid down by Otis.

5. The operation will fail if the urethra is acutely or subacutely inflamed at the time of its performance, or if such inflammation arises during the healing of the incision subsequently. The tying of a catheter into the urethra after the operation is objectionable because of the inflammatory reaction which it so frequently excites. When this occurs a greater amount of connective tissue is formed in the cicatrix and recontraction results.

6. The normal calibre of the urethra must be maintained during the healing of the incision, and this is secured by the passage of a sound of the size of the normal calibre of the urethra every second day after the operation until the wound is healed.

This requires on an average 14 days. The absence of bleeding following the use of the sound is a reliable guide for determining when the incision has healed.

Otis' Standards of Urethral Measurements.

The more important points connected with the standard of measurements laid down by Otis are these:

1. A constant relation exists between the circumference of the flaccid penis and the calibre of the urethra.

2. The penile circumference and urethral calibre vary in different individuals, consequently there is no such thing as one normal size for all urethras.

3. The average calibre of the normal urethra corresponds to No. 32 sound measured by the French scale. The penile circumference which corresponds to this is 3 1-4

inches. For every quarter of an inch of increase or decrease in the penile circumference there is a corresponding variation of 2 mm. in the calibre of the urethra.

This standard of measurements I many years ago confirmed by testing it in nearly 100 cases. I have not found a single instance in which it was not correct. Otis' standard is the only reliable, logical and accurate one that has ever been produced.

It is scarcely necessary to add that the size of the meatus is no guide to that of the urethra. The average size of the meatus is 24. A great many meatuses fall below this size, but few exceed 26. The meatus should be cut as the first step in the operation.

There remains a word to be said with regard to the best manner of dealing with the difficulties encountered in cases of impermeable stricture, and of treating those of unusual density and extent which are in the perineal part of the urethra.

With regard to the first I would say that there are but two ways which I think desirable to employ in order to trace or to make a road through an impassable stricture in the perineum.

The first of these is to perform a suprapubic cystotomy, pass a sound from within the bladder through the posterior urethra until it is arrested by the posterior face of the stricture. Next, pass another instrument from the meatus through the anterior urethra until it is arrested by the stricture. Now divide in the median line, the strictures that intervene between the points of the two instruments and the road will have been safely opened.

The second way of accomplishing this result is by exposing the membranous urethra and the anterior end of the prostatic portion of it by the classic transperineal incision which extends in a forward arch in front of the anterior margin of the anus from one ischial tuberosity to the other,

and by blunt dissection after dividing the median tendinous point of union of the structures of the perineum. The anterior bit of the prostatic urethra is then incised longitudinally, and as a rule a fine filiform bougie will readily pass from behind forward through the constricted part of the canal. Upon this instrument as a guide, the mass of connective tissues constituting the stricture is then divided.

Partial Resection of the Urethra.

The best method of treatment for the intractable strictures of the perineal portion of the canal which resist all the more usual means, is by excision of the mass of connective tissue constituting the stricture. The results obtained by this method in such cases are better than can be secured in any other way.

Summarizing what has been said it may be put thus:

1. Electrolysis and divulsion are methods of treatment which should both be abandoned.

2. Internal urethrotomy is the only method of treatment by which an important number of cures can be obtained. It is an operation involving but little danger. Its application should be restricted to strictures within the first five inches of the canal unless external perineal urethrotomy is done in combination with it.

3. For strictures of the deeper part of the canal gradual dilatation is the best form of treatment if constitutional disturbance does not arise in connection with its employment, and if the urethra for a reasonable length of time maintains the calibre to which it has been expanded by the instruments.

4. For the cases in which the strictures of the deep urethra recontract rapidly after dilatation, or those in which constitutional disturbances arise in the course of its employment, also for the resistant or impassable strictures of the deep urethra, in all of which conditions gradual dilatation is useless, external perineal urethrotomy, or internal urethro-

tomy combined with the external incision in the perineum is the safest and most efficient method of treatment.

5. External perineal urethrotomy is the only operation that should be applied in cases of stricture accompanied by urinary extravasation.

6. Resection of the strictured part of the canal should be selected in cases of intractable, very dense strictures of the perineal part of the canal.

Internal urethrotomy done under the conditions and in accordance with the rules I have stated above will yield from 50.0% to 60.0% of radical cures, and at an operative risk of death of about 1.5%.

DISCUSSION.

Dr. J. C. MUNRO, of Boston: What Dr. Watson has said upon divulsion should be printed in large letters. I have never done the operation myself, and have always condemned it as most unsurgical. External urethrotomy has always been an operation of great interest to me, and some years ago I took occasion to look up my end results, and found that the deep urethra instead of contracting had become larger in calibre than normal.

I heartily agree with Dr. Watson in his views on the permanency of the results following gradual dilatation. This method has a very limited field of usefulness and should not be placed in the same class with urethrotomy. I personally feel very grateful to Dr. Watson for what he has taught me this afternoon, and wish to express to him my thanks.

Dr. F. B. LUND, of Boston: I have been greatly interested in Dr. Watson's excellent presentation of this subject. We have at the City Hospital many cases of stricture of the urethra to treat, and we treat them all by urethrotomy; internal for the anterior strictures and external for the deep strictures. Internal urethrotomy as compared with divulsion, is a scientific and accurate procedure as compared with a violent and inaccurate one.

In a large number of external urethrotomies without a guide, which we are often compelled to perform in difficult

deep strictures at the hospital, we are practically always successful, and I have only seen one case in the hands of a colleague in which the procedure of supra-pubic cystotomy and retrograde catheterization had to be resorted to.

Many of our hospital cases belong to the class who neglect themselves after operation and consequently have to be operated upon a second or even a third time for reconstruction. It is in these recurrent cases, where the urethra is twisted and deformed by scar tissue, that the surgeon has the greatest difficulty, a difficulty however which patience will usually surmount.

I think that Dr. Watson's long experience in this class of cases enables him to speak with authority, and that his conclusions will be of great value to those privileged to hear or read his paper.

ARTICLE XXXIV.

BACTERIAL OPSONINS AND VACCINES.

By TIMOTHY LEARY, M.D.
AND
LEON S. MEDALIA, M.D.
OF BOSTON.

READ JUNE 11, 1907.

BACTERIAL OPSONINS AND VACCINES.*

INTRODUCTORY.

The following paper presents the results of fifteen months' work on bacterial opsonins and vaccines, including about 5,000 estimations of the opsonic index on 472 individuals.

The organisms studied have been: B. Tuberculosis; the pyogenic staphylococci; Streptococcus Pyogenes; Pneumococcus; Gonococcus; B. Coli; B. Proteus; B. Pyocyaneus; B. Typhosus; B. Diphtheriæ and pseudo-diphtheria bacilli.

Brief resumé of fundamental researches.

In 1884 [82] Metchnikoff brought forth his theory of phagocytosis. He concluded (1892) [83] from a study of infectious processes in animals and man, that the principal defence of the animal economy against invading bacteria lay in the power of the individual cells to envelop the bacteria with their cytoplasm and devour them by a process of intracellular digestion, analogous to that shown by free living unicellular organisms, *e. g.* amœbæ. In the higher animals the most active cells in this process are the polynuclear leucocytes, which are motile; which are drawn in inflammatory exudates to the neighborhood of the bacteria, and there exhibit their activity.

When the humoral theories of immunity (the antitoxic and lytic theories) were developed, Metchnikoff [86] still

* The expenses of this research have been borne, in part, by a fund donated through members of the profession who were interested in the work.

insisted that the activities shown by the serum against bacteria and bacterial products were due to the elaboration, by the phagocytic cells, of ferments which they set free into the blood fluids, *i. e.* extracellular ferments.

The process of phagocytosis he believed to be due, on the other hand, to direct action of the phagocytes without the intervention of any other agency.

In 1895 Denys and Leclef [23] working with antistreptococcus serum demonstrated that virulent streptococci were not engulfed by leucocytes in normal serum, while they were readily engulfed by leucocytes in antistreptococcus serum. They found that the serum acted not only on the leucocytes, but also on the bacteria, the latter uniting with the modifying agent; that the change by which cocci were prepared for phagocytosis was specific, injury or death of the bacteria not sufficing, since dead bacteria were taken up only when specific serum was added.

In 1902 Leishman [67] reported a method of measuring quantitatively the phagocytic activity of blood. He mixed in a capillary pipette equal parts of blood and a suspension of staphylococci in physiological salt solution, blew out the mixture on slides, covered with a cover glass and incubated in a moist chamber for one half hour. The cover glasses were slipped off and the films dried and stained at the end of this time. A control preparation was made with normal blood. The bacteria engulfed by a number of leucocytes were counted, and the average content per leucocyte determined. This could be compared with the average, per leucocyte, of the control blood.

In 1903 A. E. Wright and Douglas [143] published the first of a series of papers bearing upon this problem. They showed that bacteria which were mixed with washed leucocytes from indifferent sources were engulfed in almost negligible quantities by the cells, which had been freed from all serum elements by the washing. After these bacteria

had been acted upon by blood serum, however, they were readily engulfed by the same cells which previously had shown little activity toward them. They concluded that there were present in serum, substances which prepared the bacteria for phagocytosis and called these bodies "opsonins" (Latin opsonare—to prepare food for). These bodies found in normal serum were thermolabile, being destroyed in great part at 60°C for 10–15 minutes.

Based upon the method of Leishman, Wright and Douglas [143] developed a new technique for the measurement of the opsonic activities of blood sera. This consists of mixing in a capillary pipette equal parts of washed white cells, a suspension of bacteria, and blood serum. The mixture is incubated in the pipette at 37°C for, say, 15 minutes, then smeared on slides and the content of many of the cells in bacteria determined. The average number of bacteria engulfed by each cell is ascertained and this constitutes the "phagocytic index" of the blood serum used.

It was found that sera of normal individuals varied little in their power to modify bacteria for phagocytosis. It was therefore possible to use the phagocytic index of normal serum, considered as unity, for comparison with the phagocytic indices of the sera of diseased individuals. The test, then, which is always a comparative one, is carried out by making two preparations, each of which contains white cells from the same source, and portions of the same suspension of bacteria, but one of which contains normal serum, while the other contains the serum whose phagocytic index is to be compared with that of the normal, *i. e.* whose "opsonic index" is to be determined.

It was found that in local infections the opsonic index of the individual is very constantly lowered, while in general infections it is markedly variable, at one time greater, at another time less than the normal. The next step was the demonstration that vaccines consisting of suspensions of

killed bacteria could be used with success in the treatment of infections, and that their use would be followed by modifications of the opsonic index. The vaccines, since they are suspensions of bacteria, are toxic, and the immediate result of injection is a lowering of the opsonic index, the so-called negative phase, which usually persists under proper dosage for 24 hours or less. This negative phase is followed in turn by a stimulation of the immunizing machinery, resulting in an increase of opsonins in the serum—in other words, there is a rise in the opsonic index—the so-called positive phase, which may last for several days, or even a week or more. Then there is a gradual drop to normal or below.

Repeated injections properly carried out should theoretically provoke a progressive rise in the mean opsonic index, accompanied by cure of the infection.

TECHNIQUE.*

The technique of the opsonin test, as carried out by Wright, is complicated and requires a high degree of bacteriological skill, much careful attention to detail and a wide experience, before good results are obtainable. The modifications of technique which we have adopted simplify the process somewhat and make possible many estimations (30 or 40 bloods) per day by a single individual with constant good results.

The technique may be considered under seven heads, as follows:

1. Method of obtaining patient's serum.
2. Method of obtaining normal serum.
3. Method of preparing washed white corpuscles.
4. Method of preparing emulsion of bacteria.
5. Apparatus.
6. Preparations for opsonification, and staining.
7. Vaccine therapy.

* Most of the modifications of technique are due to Dr. Medalia, who also carried out most of the opsonic estimations.

1. *Patient's Serum.*

The Wright method of obtaining patient's serum consists of the collection of blood in a glass capsule with capillary ends, one of which is bent beyond a right angle with the tube. (Fig. 3.) Through capillarity the blood flows into the bent capillary end and collects in the curved portion of the tube which is held at a lower level than the drop of blood. The body of the tube is then carefully heated in a flame to expand and rarify the contained air. The straight end is quickly sealed in the flame. The contraction of the air in the body of the tube on cooling causes the blood to be drawn out of the bent capillary end which is then also sealed. The blood is then shaken down into the straight capillary end and is hung on a centrifuge to drive the clot into the conical portion of the straight end, the serum rising to the surface. The middle portion of the tube is filed and broken and the serum is ready for use. The objections to the Wright tube are its fragility, the fact that blood clots in the capillary end during filling, and that a flame must be constantly at hand for sealing purposes. The collection of blood in the tube requires some training before a good technique is developed.

We have used a tube made by drawing out ¼ inch soft glass tubing into spindles 2½ inches long with long conical ends. (Fig. 1.) The spindles are filed and broken in the middle and the conical ends sealed. Each spindle thus furnishes two blood tubes which are corked. (Fig. 2.) The similarity of these tubes to the Wright tube after it has been broken for use is indicated by the illustration. The mouths of the tubes are not annealed and the fresh surface serves to scrape the blood from the skin. The tube is simple, requires no flame and is not fragile, being readily transported by mail with little protection. The blood is collected by puncturing the ear or finger with a cutting needle (glovers No. 1) after rubbing with alcohol, a few large drops being allowed to flow into the tube.

2. *Normal Serum.*

A normal serum to serve as a basis of comparison with sera to be tested is an essential. The sera of many normal individuals may be pooled, or a single serum which is constantly compared with other normal sera may be used. The opsonic index of normal sera is almost constant, varying little or not at all from day to day. Blood is collected in the same way as in patient's serum, a larger amount being necessary, however, if many controls are to be made.

3. *Washed White Corpuscles.*

The end of the finger or the base of the nail is pricked with a glover's needle or, better, with a glass needle made by drawing out a capillary tube to extreme fineness and breaking it off. The blood pressure in the finger is intermittently increased by winding a towel or elastic ligature about it. The blood (30 to 50 drops) is collected in a tube of 3–8 inch glass, about 3 inches long, sealed at one end, and half filled with 1% sodium citrate in .85% NaCl solution. (Fig. 12.) As the blood is collected, it is thoroughly mixed with the sodium citrate solution to prevent fibrin formation. When a sufficient amount of blood has been collected, the tube is placed in a centrifuge and the blood shaken down (1200 revolutions) for 3 to 5 minutes. The corpuscles will go to the bottom of the tube, the white cells collecting in greater part in the top of the corpuscular mass, forming the corpuscular cream. The supernatant clear fluid is drawn off with a curly pipette (Fig. 10), the tube is refilled with sterile .85% NaCl solution and the corpuscular elements thoroughly mixed through the saline solution to wash the leucocytes free from serum elements. After recentrifugalization, the supernatant fluid is again pipetted off, great care being taken to remove it as completely as possible.

Animal corpuscles do not furnish as satisfactory results

as human corpuscles for routine work. Human corpuscles have therefore been constantly used in our work.

4. *Emulsions of Bacteria.*

The preparation and preservation of bacterial suspensions or emulsions is of extreme importance in the obtaining of good results. It is essential that the suspensions be homogeneous and absolutely free from clumps of bacteria. Clumps are apparently almost as readily taken up by leucocytes as single bacteria. The density of the suspensions should be such that from 2 to 5 bacteria would represent the average content of the leucocytes used in connection with normal sera. It is also essential that the bacteria should stain well.

(*a*) Tubercle Bacillus. Wright recommends the use of fresh cultures or the dry residue from the manufacture of old tuberculin. In glycerine bouillon and glycerine agar cultures the tubercle bacilli grow in long filaments forming felt-like masses, which can only be broken up by prolonged grinding in an agate mortar or between glass plates. Human tubercle bacilli from these sources are often beaded and it is a difficult matter to determine where an individual tubercle bacillus begins and ends. With poor preparations of the organisms even trained individuals will vary somewhat in their estimation of the number of bacteria contained in given leucocytes. The personal equation, therefore, enters strongly into the counting.

Our difficulties in this direction have been overcome by the use of cultures grown on Dorset's whole egg medium. On this medium tubercle bacilli grow in the form of short, solid staining rods of almost equal length. The masses of organisms are readily disintegrated without the use of the mortar, homogeneous suspensions are readily prepared, and the organisms engulfed by the leucocytes are readily counted. The method of preparation corresponds therefore very closely with that of the other bacteria, which is here given.

(*b*) Other bacteria. Bacteria are grown on agar slants or on blood serum. (Pneumococcus and Streptococcus grow best with us on Loffler's blood serum. Gonococcus on blood agar or ascitic agar.) 24° cultures are used. The water of condensation is poured or pipetted off. The tube is partly filled with sterile .85% NaCl solution and the growth is washed from the surface by churning or by the use of the platinum needle or glass rod. If a large amount of suspension is desired many cultures may be treated in the same way. If a concentrated suspension is desired the original saline may be poured from tube to tube. The suspension is next poured into a sterile test tube which has been drawn out at one point preparatory to sealing. (Fig. 8.) The tube is sealed (Fig. 13) and heated in a water bath to 60°C for one hour to kill the organisms. After killing, the sealed tube is shaken violently to break up clumped masses of bacteria, and put away 24° to allow remaining clumps to settle. The supernatant, slightly cloudy fluid is pipetted into a sterile centrifuge tube which is sealed with a rubber cap. (Fig. 7.) The phagocytic index of the emulsion with normal serum is determined and if the bacterial content is too high it may be diluted or shaken down in centrifuge.

With B. Coli and the Gonococcus, suspensions are used immediately after exposure to 60°C for 15 minutes. Emulsion is shaken down in centrifuge.

B. Coli and B. Proteus do not stain well if emulsions are heated in sealed tubes for more than 15 minutes. Work with living organisms is more satisfactory. B. Pyocyaneus emulsions do not stand prolonged heating. Even living organisms undergo bacteriolysis if long contact with normal serum is permitted, therefore living organisms are used for not more than 5 to 8 minutes.

Saline solution used.

Wright[146] recommends the use of 1.5% NaCl solution for

making suspensions of the tubercle bacillus, on the ground that spontaneous phagocytosis occurs with this organism in solutions of .85% strength. 1.5% NaCl solution, however, has the disadvantage of agglutinating T. B. It has seemed to us that spontaneous phagacytosis should be an almost constant phenomenon in mixtures (patient's and normal) which contained portions of the same suspensions of bacteria, and white cells from the same source. If so, the phenomenon is practically neglible in a comparative test where it should affect equally both mixtures used in the comparison. On the other hand agglutination results in the formation of clumps of varying size most of which are as readily engulfed as single bacteria. While churning of the bacterial suspension immediately before its use for opsonic purposes breaks up most of the agglutination clumps, it cannot be depended upon to break up all, and therefore an indeterminate error may be introduced into the estimation. For the above reasons we have used an .85% NaCl solution for tubercle as well as other suspensions. (A properly made T. B. suspension will remain free from clumps indefinitely.)

Keeping Qualities of Suspensions—

T. B. emulsion—good indefinitely.

Streptococcus emulsion—keeps for several weeks.

S. P. A. emulsion—keeps for several days.

Pneumococcus emulsion—keeps for one week.
(Heated for ½° at 60°C.)

Pseudo K. L.—keeps for two weeks.

B. Coli,
B. Proteus,
B. Pyocyaneus,
} living cultures preferable.

Gonococcus—must be used immediately after heating for 15 minutes.

5. *Apparatus.*

The standard bacteriological outfit furnishes all of the apparatus used for the test, excepting the tubes, pipettes etc., which are made from glass tubing.

Requirements—

(*a*) Thermostat—kept at 37½°C. For this purpose we use a double walled copper paraffin oven with single door.

(*b*) Oven—kept at 60° (for killing suspensions etc.) a similar paraffin oven is used.

(*c*) Centrifuge—preferably an electrical centrifuge—1200 revolutions used for ordinary work. The water motor centrifuge is said to be satisfactory. The hand centrifuge if used must be carefully handled, notably in the preparation of washed corpuscles. Jarring of the tube will favor the formation of small clots even with sodium citrate solution.

(*d*) TUBES.

1. Curly pipette—drawn from heavy glass tubing and supplied with a bulb with valve. Used for drawing off supernatant fluid from washed corpuscles, etc. (Fig. 10.)

2. Blood tubes (as described under 1). (Figs. 2 and 3.)

3. Tubes for washed white corpuscles (described under 3) (Fig. 12.).

4. Mixing pipette. Wright recommends the use of a mixing pipette made by drawing out one end of a piece of large glass tubing and fitting a teat such as is used in a medicine dropper over the end. We have found it simpler to use mixing pipettes made by drawing out ⅜ in. glass tubing into fine capillaries with a short spindle between the capillary portions. (Fig. 4.) The tube is broken off close to one side of each spindle—thus furnishing a tube with a long capillary arm at the end of which is a dilated portion (spindle) over the other end of which is fitted a small teat such as is supplied with tubed glycerinated vaccine. The principal advantages of this tube over Wright's are that it is more readily made in quantities—the teat is more rapidly fitted—the teat is small and therefore more readily controlled—the whole pipette is much smaller and less awkward to handle. (Fig. 5.)

A modification of this tube is shown in Fig. 6. The end of teat in this case is thrust into the glass tubing, thus making a permanent joint and so compressing the rubber outlet of the teat that a column of fluid in the attached capillary tube is very readily controlled. Capillary tubes only need be drawn out for this modification. The connection is made with a piece of capillary rubber tubing, such as is used for covering spectacle frames.

(*e*) No. 00 emery paper (watchmaker's) is used for the cleaning of slides. The emery scratches the polished surface of the glass and blood films spread more evenly[134].

(*f*) Heavy test tubes drawn out for sealing (used for killing emulsions) Fig. 8.

(*g*) A spreader for films is made by cutting off the corners of a glass slide and rubbing the edge with emery paper.

(*h*) Centrifuge tubes covered with rubber caps for holding emulsion. (Fig. 7.)

(*i*) Holders for vaccine—individual dose holders may be made from glass tubing, or bottles sealed with rubber caps may be used (Whitall, Tatum & Co. 2 dram amber French square) capped with rubber (Bausch & Lomb 18 m.m. caps). (Fig. 11.)

(*j*) Tubes for churning bacteria—heavy glass tubing drawn to a fine capillary end with heavy walls—and supplied with a rubber bulb without valve. (Fig. 9.)

(*k*) The following sterile solutions should be kept in flasks with siphon connection:

.85% NaCl solution.
.85% NaCl solution with 1% sodium citrate (for defibrinating purposes).
.85% NaCl solution with .25% carbolic acid (for diluting vaccines).

(*l*) Glass cell slides for holding suspensions of bacteria.

(*m*) Glass pencils. (A. W. Faber, blue are best.) Many glass pencils make marks which wash off in solutions.

(*n*) Paper cut in pieces 2½x2 inches, for labelling opsonizing pipettes.

6. *Opsonification.*

Bloods to be tested are kept in the ice-chest until used. When the operator is ready, blood tubes are removed and shaken down in centrifuge. Washed corpuscles are prepared as described. A mixing pipette is drawn out in the flame and the sharp sterile end is thrust through the rubber cap of a sedimentation tube holding bacterial suspension. Enough of the suspension is withdrawn to satisfy requirements and placed in a cell slide. This is repeated for each emulsion desired, a new mixing pipette being drawn out for each suspension. Each suspension is thoroughly churned through a fine capillary tube against the glass of the cell slide to break up any clumps.

The operator has now before him blood (serum) in tubes, washed white cells and a suspension of bacteria in a cell slide. The cover of a paper slide box, with numbered perforated holes, makes a satisfactory rack for blood tubes.

With a glass pencil a mark is made ¼ to ½ inch from end of capillary of mixing pipette. This is to mark the unit of measurement. One volume of washed white cells is drawn into capillary, then a bubble of air—then one volume of bacterial suspension—another air bubble, and finally one volume of the serum to be tested. The contents of the capillary tube are now expelled on to the surface of a glass slide and drawn back and forth with capillary tube until thoroughly mixed. This mixture is finally drawn well up into the capillary tube, care being taken to exclude air bubbles, which interfere with phagocytosis. The tube is then sealed in the flame, and while still hot the end is thrust in and out of a piece of paper which serves for the labelling of the specimen. Label is marked with name of patient, bacterium and time.

For each bacterium used a control preparation is made with normal serum. It should be kept constantly in mind that the test is a comparative one and therefore the patient's serum and the normal (control) serum mixtures should be prepared and incubated under exactly similar conditions, notably as to time.

After incubation for 15 minutes the tube is withdrawn from thermostat. Two glass slides having been prepared with emery paper, the tip of the capillary tube is broken off and the contents are churned back and forth on one of the slides. This remixes the materials which have sedimented in capillary and breaks up leucocytic clumps. One-half of the material is then deposited on each slide about 1 inch from end. With a glass spreader held at an angle of 40° the mixture is smeared over the surface. The leucocytes, which are larger bodies than the reds, are held back by the spreader and roll out over its edge. Leucocytes are found therefore in largest numbers along the edge of film and at end.

FIXING.

Smears are labelled with a glass pencil and are dropped into glass slide-boxes filled with saturated aqueous solution of corrosive sublimate. (The sublimate is kept in super-saturated solution and the supernatant fluid is used.) After exposure of from 1 to 2 minutes the corrosive is poured off and slides washed thoroughly in running water.

STAINING.

The slides having been fixed and washed are stained as follows:

(*a*) Bacteria other than T. B.

Most writers recommend J. H. Wright's or Leishman's stains. These stains are not easily handled with large numbers of slides, tend to deposit on films, and the staining of the leucocytic granules interferes with the rapid recognition

of the contained bacteria during the process of counting. Carbol-thionin is expensive and not easily handled. We have used with the greatest satisfaction the following simple stain:

Sat. alc. meth. blue	25.
.3% aqueous sol. sod. carb.	75.

Stain from one to several minutes.

Wash off quickly with water.

Dry quickly.

The stain keeps indefinitely if sodium carbonate is added from time to time to overcome the acidity, which probably is due to the acid corrosive sublimate.

(*b*) Tubercle Bacilli.

Fix as above. Stain with carbol-fuchsin, steaming for 1 minute. Wash off excess of stain in water.

Place in 2% H_2SO_4 solution 20 seconds.

Wash in water.

Counterstain with sodium carbonate blue as above.

Wash carefully.

Dry and mount.

The use of acetic acid to dissolve the red cells, we have not found to be an advantage. The sulphuric acid in properly made films destroys these cells in large part. As a matter of fact the continuous film of red cells is a distinct advantage on account of the readiness with which the focus is held.

COUNTING.

The counting of bacteria is carried out with a mechanical stage to ensure that ground is not recovered. As noted, the leucocytes will be found in greatest number along edges and at end of film. Only isolated leucocytes should be counted. Cells in relation to clumped bacteria should be ignored. Good fields will show from one to five cells—no more. Representative portions of both slides should be examined.

The content of 50 cells in bacteria having been determined, the average content per leucocyte is estimated. This constitutes what is known as the phagocytic index. The opsonic index is determined by comparing the phagocytic index of the patient's serum with the phagocytic index of the normal (control) serum, example:

1		2	
Washed white cells. Suspension of T. B. *Normal serum*	*aa.*	Same washed white cells. Same suspension of T. B. *Patient's serum.*	*aa.*

Incubated at 37°C for 15 minutes.

50 polynuclear leucocytes.	50 polynuclear leucocytes.
engulf 150 T.B.	engulf 100 T.B.
Phagocytic index=50)150(3 150	Phagocytic index=50)100(2 100

The phagocytic index of the patient's serum is $\frac{2}{3}$ that of the normal serum, which is taken as unity, and its opsonic index is therefore indicated as .67.

VACCINES.

Cultures are prepared as for the making of bacterial emulsions. Growths for 24 hours or less are best for this purpose. With serum the water of condensation is pipetted off. There is then added to the first tube a small quantity (5 to 10 c.c.) of .85% NaCl solution. The growth is separated from the medium by a platinum needle or a bent glass rod. Churning facilitates the emulsion of the organisms. The fluid is then poured into a second culture and the process repeated. As many cultures may be used as seems desirable. After thorough emulsification the number of bacteria present in the emulsion is estimated. With the streptococcus the emulsion should be poured into a test tube which is drawn out in the flame and then sealed. Violent shaking is necessary in order to break up the chains. If this is not done the estimation of the number of bacteria is unsatisfactory.

The emulsion being homogeneous, equal quantities of emulsion and blood are measured in a mixing pipette with 2 or 3 volumes NaCl solution, the technique corresponding to that used for the opsonin test[178]. The material mixed thoroughly on a glass surface is then smeared on slides and stained by the technique described. With an eyepiece diaphragm to limit the field (made by punching or burning a hole in a piece of cardboard) the content of many fields in bacteria and cells is determined. At least 300 red cells should be counted. Normal blood contains about 5,000,000 cells per cubic m.m., or, 5,000,000,000 cells per cubic centimeter. The ratio of red cells to bacteria in the fields counted, related to 5,000,000,000, will furnish an estimate of the number of bacteria present per cubic centimeter. Number of red cells: number of bacteria :: 5,000,000,000 : x. Example:

The fields counted show 735 red cells and 1044 bacteria. 755) 1044 (1.42 x 5,000,000,000 = 7,100,000,000 bacteria per c.c. of emulsion.

The suspensions are killed in sealed tubes at 60° C for ¾ to one hour. After heating, tubes are unsealed and large portions of emulsion are planted on culture media and incubated in order to make sure that bacteria are dead. The emulsions are in the meantime kept in the ice-chest. When the sterility of the emulsion is established it is diluted with .85% NaCl solution containing .25% carbolic acid, and stored in sterile receptacles in the ice chest.

Standard vaccines are put up with the following content: Staph. Py. Aureus – 500,000,000 per ½ cubic centimeter (7½ minims): 250,000,000 per ½ c.c.: 50,000,000 per ½ c.c. (for children).

Streptococcus – 25,000,000 per ½ c.c.; 50,000,000 per ½ c.c.

Pneumococcus—50,000,000 per ½ c.c.; 100,000,000 per ½ c.c.

B. Coli – 50,000,000 per ½ c.c.; 100,000,000 per ½ c.c.

B. Proteus – 100,000,000 and 200,000,000 per ½ c.c.

B. Pyocyaneus – 100,000,000 and 200,000,000 per ½ c.c.

Gonococcus – 25,000,000, 50,000,000 and 100,000,000 per ½ c.c.

As a vaccine in Tuberculosis, Koch's Tuberculin T. R. is used. This is diluted with the saline-carbolic solution so that each ½ c.c. contains 1–1000 mg. of dried powdered tubercle bacilli.

Our standard vaccines are kept in 2 dram amber French square bottles sealed with a rubber cap. This bottle holds 10 c.c. The bottles are given prolonged boiling before use and the caps are kept in 5% carbolic solution from which they are taken directly, squeezed to remove carbolic, and fitted on bottles. For use the cap is covered with 5% carbolic solution and allowed to stand for a few moments. The needle of a sterile hypodermic syringe is thrust through cap, the desired quantity of vaccine removed, and the needle withdrawn. Unless needle is thrust through to the hub the hole made will reseal itself.

Autogenous vaccines, *i. e.*, vaccines made with bacteria obtained from the patient's lesions, are desirable or necessary in many infections, notably those due to the Streptococcus and B. Coli. Where pure cultures are obtained a vaccine may be produced in less than 24°. B. Coli vaccines may be obtained in 6 to 8 hours. Where the immediate exhibition of an autogenous vaccine is necessary, as for instance in B. Coli peritonitis or in puerperal sepsis, (Streptococcus) the vaccine is used without waiting for the establishment of sterility through cultures. We have never obtained growths after exposing an emulsion in *sealed* tubes for one hour at 60°C.

OPSONINS NECESSARY FOR PHAGOCYTOSIS.

The Metchnikoff school have taken the position that phagocytosis was a purely cellular function. Lohlein [71] says,

"Phagocytosis is a cellular act which can operate without the intervention of substances contained in the serum." The experiments of Lohlein which led to this conclusion consisted in washing leucocytes free from body fluids in saline solution (peritoneal exudate, produced in g. pigs by the injection of bouillon, furnished the leucocytes used); adding the washed leucocytes to suspensions of bacteria (no details) and incubating for 2 hours or less. He claimed to have obtained with certain strains of streptococci and B. Coli–B. Anthracis and Sp. Choleræ Asiaticæ as marked phagocytosis in 2 hours as was obtainable with Wright's mixtures of serum, washed whites and bacteria in 15 minutes. No exact details are furnished, however, of the number of bacteria picked up.

Our results from similar experiments are illustrated in a following table. (Table I).

In a later communication Lohlein [72] admits that serum bodies are essential for the phagocytosis of most bacteria. His earlier experiences in which some bacteria were picked up readily without serum action lead him to believe, however, that there are exceptions to the rule. Tunnicliff and Davis (quoted by Hektoen [47]) found that fusiform bacilli and spirilla and influenza-like organisms were taken up by washed leucocytes in saline solutions (to a less degree, however, than in serum).

Wright [146] recognized that spontaneous phagocytosis occurred when leucocytes and bacteria in saline solutions of low concentration were brought together (see Table I). By the use of 1.5% NaCl solution spontaneous phagocytosis could be practically eliminated. Wright therefore recommends that 1.5% NaCl solution be used for opsonic estimations, particularly with the tubercle bacillus, since spontaneous phagocytosis is shown most markedly in the presence of this organism. (With saline solution of this strength, however, a new factor is introduced. Tubercle

TABLE I.

	I.	II.	III.	IV.	V.	VI.	VII.	VIII.
	S. P. AUREUS. HEAVY.	S. P. AUREUS. HEAVY.	S. P. AUREUS. LIGHT.	S. P. AUREUS. LIGHT.	S. P. AUREUS. LIGHT.	B. COLI. I.	B. COLI. II.	B. COLI. III.
Phagocytic Index. Normal Serum (control)	24.78	11.06	3.14	2.80	2.88	7.14	6.26	5.78
Phagocytic Index. Saline (control)	1.64	.20	.12	.26	.20	.44	.74	.26
Phagocytic Index. Saline—2° incubation	1.96	.36	.22	.20	.18	.70	.86	.30
	Incubation 15 min.	Incubation 15 min.	Incubation 15 min.	Incubation 15 min.	Incubation 2 hours.	Incubation 15 min.	Incubation 15 min.	Incubation 15 min.

While it is notable that the phagocytic indices of the saline, which has been digested with leucocytes for two hours, are higher than that of the control saline, it will be seen that where light bacterial suspensions, such as are used in opsonic estimations, have been mixed with saline, the indices are too low to affect seriously results, even after 2° incubation.

bacilli in 1.5% NaCl solution undergo rapid agglutination in clumps of varying size, and it is a difficult matter to free tubercle emulsions from such clumps. See technique).

Spontaneous phagocytosis occurs then with exceptional bacteria, which are readily picked up by leucocytes in saline solution, and also to a very limited extent with the bacteria ordinarily used for opsonic estimations when NaCl solutions of .85% or weaker strengths are used.

Another conception of the Metchnikoff school is that leucocytes are the producers of opsonins.

The experiments which are charted in Table I were carried out, (a) to determine the degree of spontaneous phagocytosis which would be shown under the conditions which apply in the ordinary process of opsonification; (b) to determine whether leucocytes digested for 2 hours (the optimum period of Lohlein's [72] experiments) with .85% NaCl solution would give up to the saline solution substances capable of influencing phagocytosis; (c) in one case (column V) to determine whether incubation for two hours with saline solution or leucocytic extract would increase markedly the phagocytosis. Staphylococci and three strains of B. Coli were used, since Lohlein found that these organisms were readily picked up by leucocytes in saline solutions. The suspensions which were used in experiments shown in columns III, IV, V, VI, VII and VIII were standard suspensions such as are used in ordinary opsonic tests. The suspensions used in experiments whose results are shown in columns I and II were much heavier than those ordinarily used.

The first row of figures shows the phagocytic indices of serum, incubated with bacterial suspension and washed whites for 15 minutes at 37°C, as in the application of the ordinary opsonic test.

The second row of figures shows the results of incubation of .85% saline solution with bacterial suspension and

washed whites for 15 minutes. (In column V the results of incubation for two hours with the same suspension used in experiments shown in column IV are detailed.)

For the results shown in the third row of figures a leucocytic extract was prepared by digesting washed white cells and .85% NaCl solution for two hours at 37°C. The mixture was then centrifugalized and the supernatant saline was mixed with fresh washed white cells and the suspension of bacteria. Incubation for 15 minutes, except in the experiments whose results are shown in column V.

Possible sources of error in estimation of the opsonic index are indicated in Table II.

The first column of figures shows the phagocytic index of saline solution obtained from the first washing of leucocytes—together with first wash leucocytes—the second column the index of saline solution obtained from the second washing of leucocytes, together with second wash leucocytes. The third column shows the result of the use of leucocytes 24° old. The same suspension of S. P. Aureus was used in these estimations.

It will be noted that in column 1 the indices are high, due undoubtedly to the failure to remove opsonin left adherent to the leucocytes or suspended in the saline solution, on account of the incomplete washing. Column 3 shows the diminution in the activity of leucocytes, due to age.

LEUCOCYTES.

Wright[143] and others have shown that it is an almost indifferent matter whether leucocytes from one healthy animal or from another are used in opsonic estimations. While some variation is shown in the ability of cells to pick up sensitized bacteria it is not great enough to be of serious moment. Reudiger found that human leucocytes from a case of acute nephritis were less active than those of normal individuals.

Apart from the exceptions noted, the concensus of scientific medical opinion, with which our results agree, is that bacteria are engulfed by leucocytes only after their preparation by certain serum bodies, which Wright [143] has tentatively named "opsonins."

THE CHARACTER OF OPSONINS.

Before discussing this question, a distinction which apparently exists between the opsonins found in the serum of a normal individual and those which are found in individuals who are undergoing inoculation with bacterial products, should be considered.

Wright [135] has shown that in the progress of a local disease, *e. g.*, tuberculosis of bone—operation, massage or physical exercise will result in the production of a typical opsonic vaccine curve, *i. e.* a negative phase, followed by a positive phase with later subsidence. His explanation of this occurrence is that the operative or manipulative measures set free from the patient's local focus of infection bacterial products which affect the immunizing machinery, just as would the injection of a bacterial vaccine.

In immune sera Wright and Douglas, [143] Dean [30] and Hektoen and Reudiger [62] demonstrated that heating for limited periods to 60°C did not destroy all of the opsonins, while normal serum similarly heated lost almost completely its opsonic power (with few exceptions). Further investigations have indicated that there are other points of difference between what may be called "normal" and "immune" opsonins.

NORMAL OPSONINS.

Normal opsonins, in addition to being thus thermolabile, are destroyed rapidly if serum is exposed to sunlight, or if kept at body temperature (Wright [143]); disappear more slowly if serum is kept at room temperature, and are preserved indefinitely at 0°C (Horton [55]). The opsonic activ-

TABLE II.

15 Min. Incubation—Suspension S. P. A.	Leucocytes After 1st Washing.	Leucocytes After 2d Washing.	Leucocytes 24 Hours Old.
Phagocytic Index of Serum Control	4.88	4.12	3.88
Saline from Leucocytes	1.30 Saline—1st wash.	.80 Saline—2d wash.	.24 Saline—2d wash.

TABLE III.

	B. Tuberculosis.		Pneumococcus.	S. P. Aureus.		B. Typhosus.	B. Coli.	
	Pooled Normal (Human.)	Rabbit Serum.	Rabbit Serum.	Pooled Normal.	Normal.	Normal.	Pooled Normal.	Rabbit Serum.
Controls	1.30	2.22	11.76	1.54	(5.32)	(1.40)	1.56	3.42
Serum sat. with T. B.	*.58*	*.66*	12.54	1.80			.94	2.66
Serum sat. with Pneumococcus .		1.44	*.60*					2.30
Serum sat. with S. P. A. . . .	1.28			*.84*	(*.28*)	(1.24)	.94	
Serum sat. with B. Typhosus .					(.92)	(*.28*)		
Serum sat. with B. Coli . . .	1.06	1.22	1.76	1.10			*.34*	*1.20*

ity of normal serum is progressively lessened as the serum is diluted (Hektoen & Reudiger[52]). The opsonic power of normal serum is inhibited or destroyed by many chemical agents, such as isotonic solutions of the chlorides of Ba, Ca, Sr. and Mg.—the carbonates of Na, K_2SO_4 and notably by lactic acid, formalin, and other agents which are known to be capable of producing negative chemotaxis, *i. e.* are able to repel leucocytes (Hektoen & Reudiger[52]).

Bulloch and Atkin[19] have shown that saturation of normal serum with a bacterium for a sufficient length of time will remove completely the opsonins for that bacterium. Muir and Martin[93] have shown that the opsonins of normal serum are absorbed (diverted) by combinations of receptor and immune body for bacteria, or red cells, and by precipitins for serum albumin.

The specificity of normal opsonin has been studied by Bulloch & Western,[21] Rosenow,[106] and Muir & Martin.[93]

Our own results from more complete series of estimations are shown in the subjoined tables. In each case the serum was digested with a suspension of bacteria (1/20th of amount of 1,000,000,000 per c.c. suspension) for 45 minutes at 37°C. No attempt was made to establish complete saturation. At the end of this period the mixture of bacteria and serum was centrifugalized for 30 minutes or more at 2800 revolutions (ordinary centrifuge arm). The supernatant fluid thus freed from bacteria was mixed with washed whites and a standard suspension of another bacterium, and incubated for 15 minutes at 37°C. At the end of this time the bacteria engulfed by 50 white cells were counted and the average (phagocytic index) estimated.

Table III. illustrates the results obtained in three series of observations, one with a single normal human serum (normal), one with pooled normal human serum, and one with rabbit serum. The controls show the phagocytic in-

dices of the sera before saturation. The comparative specificity of the opsonins is well illustrated, although particularly with sera saturated with B. Coli, the abstraction of opsonin for other bacteria is well marked.

Table IV. illustrates the results obtained with the serum of apparently normal rabbits and guinea-pigs. As in the previous table, the comparative specificity of the normal opsonins is evident, although in practically every case there is a loss of opsonic power, toward all of the bacteria used, after the digestion with bacterial suspension.

The results of Bulloch & Western and Muir & Martin, obtained with a technique corresponding to ours, and of Rosenow, who digested mixtures of bacteria and serum for 12 or more hours, then filtered to free serum of bacteria, harmonize very closely with our own.

IMMUNE OPSONINS.

The thermo-stability of immune opsonins has been established by Savtchenko,[114] Dean,[30] Leïshman,[68] Neufeld and Rimpau,[95] Muir & Martin,[93] and others. The ability of immune sera to prepare *virulent* bacteria for phagocytosis has been repeatedly demonstrated since the paper of Denys & Leclef[33] in 1895, in which the serum of animals immunized to the streptococcus was shown to have this power. That the action is absolutely specific; *i. e.*, will not be exhibited against any other bacterium than that with which the animal was prepared, was shown by Neufeld & Rimpau,[95] Muir & Martin,[91] and others. Muir & Martin[91] further find that immune opsonins cannot be absorbed by mixtures of receptor and immune body. This is in marked contrast to the reaction of normal opsonins under similar conditions.

Neufeld & Rimpau in 1904,[95] and independently of Wright, found that anti-streptococcus serum mixed with virulent streptococci so modified the bacteria that they were

TABLE IV.

	Streptococcus.				Staph. Py. Aureus.				Pneumococcus.		B. Coli.	
	Rab. No. 1.	Rab. No. 2.	G. Pig. No. 1.	G. Pig. No. 2.	Rab. No. 1.	Rab. No. 2.	G. Pig. No. 1.	G. Pig. No. 2.	Rab. No. 1.	G. Pig No. 1.	Rab. No. 1.	Rab. No. 2.
Control-Phagocytic Index Serum .	2.84	4.42	4.22	4.68	3.34	4.26	7.56	6.42	2.56	2.20	1.86	2.74
Serum saturated with Pneumococci .	2.20		3.18		2.74		4.52		*.52*	*.60*	1.45	
Serum saturated with Streptococci .	*1.18*	*.58*	*1.08*	*1.80*	3.76	4.84	5.14	4.82	2.	1.68	1.40	2.42
Serum saturated with S. P. Aureus .	2.26	3.28	2.80	3.24	*1.68*	*1.42*	*1.42*	*2.46*	2.	1.88	2.48	1.92
Serum saturated with B. Coli . . .	2.66	1.95			2.52	2.96			2.32		*.78*	*.90*

readily engulfed by white cells. On the other hand, leucocytes treated with the anti-streptococcus seru for 15 minutes at 37°C, then washed free from serum elements, showed no phagocytic activity after reincubation with a mixture of virulent streptococci and normal serum. The serum, therefore, sensitized the bacteria, and not the leucocytes. The writers gave to the bodies in immune serum which prepared bacteria for phagocytosis the name of bacteriotropins, *i. e.* bodies which in contrast to bacteriolysins are directed against the virulence-carrying receptors of bacteria. They concluded that a new group of anti-sera must be added to those already known—the antitoxic and lytic—and suggested the name "bacteriotropic."

Wright and Reid[146] have shown that immune opsonin may be completely absorbed from sera by saturation with suspensions of their specific bacteria; that the activity of immune opsonin is lessened progressively with the dilution of the serum; that the opsonin found in heated serum is destroyed by heat when the serum is sufficiently diluted; that the immune opsonin, while apparently thermostable, is quickly destroyed by heating, after exposure to the direct action of the sun, *i. e.* is heliolabile.

Wright, from these findings, argues for the identity of the opsonins found in normal and immune sera, claiming also that the conception that the immune opsonins are new bodies, which arise only under conditions of immunization, is in contravention of the fundamental laws of immunity. He says: "We submit that it is erroneous to conceive of the normal organism as differing from the immunized organism in a qualitative manner. Rather does not the theory of Ehrlich brilliantly teach that in immunization we are never building up from an absolute level of non-resistance, but always building on a foundation which is already laid—calling into existence in increased quantity and conveying into the blood only such chemical agents as exist pre-formed in the body?"

The question, then, of the difference of normal and immune opsonins is still a debatable one, most observers, however, holding that the differences are qualitative as well as quantitative.

RELATION OF NORMAL OPSONINS TO KNOWN ANTIBODIES.

1. Resemblance to complements.

Normal opsonins, like complements, are destroyed by heat, sunlight and standing.

Normal opsonins, like complements, may be bound by various salt solutions, and so prevented from acting on bacteria. (Hektoen.[52])

Normal opsonins, like complements, are not absolutely specific; *i. e.*, have certain communal relations. (See charts on specificity.)

Normal opsonins, like complements, may be absorbed by lytic mixtures; *i. e.*, red cells + antiserum in which the complement has been destroyed by heating — and bacteria + immune serum, similarly heated to remove complement. The fact that lytic action does not follow opsonification does not necessarily modify the conception that opsonins are complement-like bodies, since Muir and Browning[61] have shown that the union of complements with cells and immune bodies is not followed by lysis in some cases.

On the other hand, bacteria which have been acted upon by the opsonins of normal serum are modified only in becoming more phagocytable. That they are not only not dissolved, but are not killed, by the action of the serum alone, before phagocytosis, has been shown by many culture experiments.

According to Lohlein,[72] normal opsonins differ from complements in that they unite with bacteria at 0°C, a temperature at which complements are inactive.

Complements, while in themselves very simple bodies, require the intervention of amboceptor or immune body in

order to complete their action. The indications from many experiments are that the union of normal opsonins with bacteria is a simple process analagous to that shown by agglutinins and precipitins, and corresponding in character to the union of toxins and antitoxins. Hektoen[62] believed that by heating bacteria which had been opsonized by exposure to normal serum, he rendered the organisms insusceptible to phagocytosis even when they were later exposed to the action of unheated normal serum. His conclusions were that opsonins, like agglutinins and precipitins, were composed of haptophore and zymophore (opsoniphore) groups; that the heating of the bacteria destroyed the opsoniphore group, leaving the thermostable haptophore group attached to the cell; and that the haptophores attached to the bacterial cells prevented the union with these cells of the haptophores of the opsonins of fresh normal serum. Experiments which we are carrying out, but which are not completed, apparently do not confirm Hektoen's observation.

RELATION TO AGGLUTININS AND AMBOCEPTORS.

Lohlein[72] believes from his researches that the opsonins most closely resemble agglutinins.

The distinctions between opsonins and lytic amboceptors and agglutinins are clear and are summarized as follows, by Hektoen[47]:

1. Normal serum may possess lytic power, but not opsonic, and vice versa.

2. Immunization may give rise to opsonic substances, but not to lytic or agglutinating.

3. Heat may destroy opsonic power, while the lytic amboceptors remain intact.

RELATION OF IMMUNE OPSONINS TO ANTI-ENDOTOXINS.

Immune opsonins are unlike normal opsonins in that they resist exposure to 60°C for prolonged periods, are more

absolutely specific, are not diverted by mixtures of receptor and immune body, and in that they bring about phagocytosis of virulent bacteria. They are therefore, according to most observers, distinct bodies and dissimilar to the opsonins of normal serum.

It appears to us that these bodies (immune opsonins) have a very definite and close relation to the antiendotoxins first described by Besredka[8], as will be seen from the following:

In natural infections, the phagocytic action of the body cells is exhibited against bacteria which owe their power to infect to the evolution of endotoxins. In laboratory experiments it is true that phagocytosis will be exhibited also toward bacteria, such as the diphtheria bacillus, which owe their infecting power to the evolution of extra-cellular toxins; but in natural infections with these bacteria, the important substances produced by the tissues in opposing the infection are anti-toxins, phagocytosis being a secondary phenomenon, if exhibited at all. The close relation of phagocytosis to endotoxin-producing bacteria is of itself significant.

Virulent bacteria are insusceptible to phagocytosis after being exposed to the action of the opsonins of normal serum. Rosenow[105] has extracted from virulent pneumococci, by heating to 60°C after prolonged action of .85% saline solution, toxic bodies which he calls virulins. After the extraction of these bodies the pneumococci appear to have lost their resistance to opsonification by normal serum, although observations are inexact because of the poor staining of the pneumococci, which are partially disintegrated. That the heating of virulent bacteria without extraction is of itself not sufficient to destroy their anti-opsonic properties has been shown by Hektoen[61] and others. It is evident, then, that substances which are extractable from virulent bacteria after heating, and are themselves toxic, are re-

sponsible for the resistance of these organisms to opsonification by normal sera. (The streptococcus and other bacteria produce in addition to the specific anti-opsonic substances, bodies which cause the destruction of leucocytes, *i. e.*, leucotoxins. The pneumococcus does not apparently produce leucotoxins. Reudiger.[111])

Besredka[6] succeeded in extracting from killed B. Typhosus, B. Pestis and B. Dysenteriæ soluble endotoxins, whose toxicity he measured on animals. By the intravenous injection of killed cultures of the organisms (from which soluble endotoxins were extractable) into animals, he was able to bring about the production of serum substances which neutralized the toxicity of the soluble endotoxins. Measured quantities, expressed in terms of minimal lethal doses of endotoxins for g. pigs, were neutralized by measured quantities of the antiserum just as is the case with diphtheria or tetanus antitoxin *vis a vis* its corresponding toxin. Besredka[5] concluded that serum substances capable of neutralizing endotoxins were properly anti-endotoxins, although bacteriological tradition has always taught that the formation of these bodies did not occur.

To recapitulate:

Analogies between endo-toxins and the substances which prevent the phagocytosis of virulent bacteria.

Endotoxins. (Besredka.)	Virulins. (Rosenow.)
Thermostable.	Thermostable.
Action of *specific* immune serum neutralizes.	Action of *specific* immune serum permits phagocytosis.
Extractable from bacteria by prolonged action of saline solution and heat.	Same.
Bacteria remaining after extraction are pratically non-toxic.	Bacteria remaining after extraction are apparently phagocytable.
Extract is toxic.	Extract is toxic. Its addition to avirulent bacteria causes them to resist phagocytosis.

Virulence of bacteria dependent upon the possession of these bodies.	Resistance to phagocytosis varies *pari passu* with variations in virulence.

The analogies between endotoxins and the substances which prevent the phagocytosis of virulent bacteria are so close that it is reasonable to presume that we are dealing with the same body in each case. If this is true, it would follow that the same antibodies would be produced following injection into animals. A very careful reading of the literature from this viewpoint develops no objections to this hypothesis.

Analogies between anti-endotoxins and immune opsonins:

Anti-endotoxin.	Immune opsonin.
Specific anti-body produced by tissues in response to the action of bacteria or endotoxin.	Same.
Found in large quantities in immune serum; in small quantities in normal serum (? of previous infection or subinfection)	Same.
Neutralizes endotoxins of specific virulent bacterium.	Favors phagocytosis of specific virulent bacterium.

From the standpoint of bacterial infection and immunity the so-called immune opsonins are of paramount importance in phagocytosis. In infections we are dealing always with virulent bacteria which normal opsonins cannot modify. Under conditions of infection or vaccination it is the specific immune opsonin which is increased (Bulloch & Western[21] —Muir & Martin[91]) and it is the activity of this body which is responsible for the healing of infection and the development of immunity.

The conception that immune opsonins are anti-endotoxins capable of neutralizing specifically the endotoxins of virulent bacteria, which, deprived of their weapons, thus fall easy prey to phagocytes, would simplify greatly our understanding of the process of phagocytosis. Such a conception

would account for the simplicity of the process of opsonification since antiendotoxins probably correspond in combining character to the known antitoxins, whose method of union with toxins has been shown by Ehrlich to be a simple one.

Whether the action of normal opsonins on avirulent bacteria is due to the presence in serum of substances less specific and capable of extracting or neutralizing mildly toxic substances from these organisms must be left an open question. The fact that some bacteria are phagocytable in saline solutions alone (Lohlein, Tunnicliff & Davis), while a second avirulent group (including virulent organisms which have lost virulence after growth on artificial media) requires the action of normal serum, and a third (virulent) group requires the action of specific immune serum in order to be phagocytable would suggest that this is probably true. It is our intention to do further work along these lines.

Part II.

Clinical Application of Opsonic Test.

A. *Its Application in Diagnosis:*

Wright's demonstration that low opsonic indices to a bacterium were associated with local infection by that bacterium, while markedly variable indices were associated with general infection, led to the applications of these findings as a means of diagnosis.

Wright's [147] tenets which he laid down specifically for the diagnostic application of the opsonic test in tuberculosis are apparently applicable in other conditions. They are as follows:

a. "Where a series of measurements of the opsonic power of the blood reveals a persistently low opsonic power with respect to the tubercle bacillus, it may be inferred, in the

case where there is evidence of a localized bacterial infection, suggesting tuberculosis, that the infection in question is tubercular in character."

b. "Where repeated examinations reveal a persistently normal opsonic power with respect to the tubercle bacillus, the diagnosis of tubercle may with probability be excluded."

c. "Where there is revealed by a series of blood examination a constantly fluctuating opsonic index the presence of active tuberculosis may be inferred."

From an isolated blood examination the following conclusions may be arrived at (Wright):

d. If the tuberculo-opsonic power is low and there is evidence of localized infection or constitutional disturbance it may be inferred that there is with the former a local—with the latter a general tubercular infection.

e. If the tuberculo-opsonic power is high, we are dealing with a systemic tubercular infection which is active or has recently been active.

f. If the power is normal no inferences should be drawn.

We have preferred to obtain a second or third blood rather than draw inferences from a single blood.

The occurrence in immune serum of immune opsonins capable of resisting exposure to 60°C for 10 minutes has been used to a limited extent by Wright as the basis of a diagnostic test. If blood after heating to 60°C retains any considerable part of its opsonic power, it may be concluded that immune opsonins arising either from auto-vaccination or from artificial vaccination are present. The testing of heated serum has had only a limited application and has furnished somewhat contradictory results. It has not been satisfactory in a limited application with us.

Mention has been made of the negative phase, which follows the injection of vaccines. A similar negative phase will follow in individuals with local infections, on operation, massage, physical exercise or the use of X rays.

(See record of post operative results.) In normal individuals the injection of vaccines, unless in overdoses, is not followed by a negative phase, but usually by an immediate rise in the opsonic index to the bacterium used. The occurence of a negative phase following vaccination of infected individuals has been given much attention by English observers (Lawson & Stewart[66]). As a diagnostic test in tuberculosis they claim for it 100% of successes as compared with a much lower percentage of clinical reactions to inoculations of old tuberculin.

PRACTICAL APPLICATION OF METHOD.

Before applying opsonic findings diagnostically it is essential that an appreciation of what constitute "normal limits," *i. e.*, the limits within which the opsonic index of the blood serum of normal individuals may vary, should be had. Wright fixed these limits for tests with the tubercle bacillus at .8 and 1.2. It may be said that in our practical work we have fixed the "normal limits" to all other bacteria within these bounds.

Where possible two or three specimens of blood are obtained at intervals of 48 hours or more. This is particularly true in cryptogenetic proccsses where cultures are not obtainable. Where cultures are obtained, but show a mixed growth of many varieties of bacteria, it is often true that the important infecting agent may be so overgrown by other organisms that its relative importance is overlooked. In fact, in some cases the important infecting agent may not appear on cultures taken with the ordinary precautions from a clinical standpoint (see introduction to chapter on uterine sepsis).

In processes in which the tubercle bacillus is suspected of being the important infecting agent direct, cultivation of this organism is often difficult or impossible, particularly in lesions on surfaces exposed to contamination. Even from

uncontaminated sources the cultivation of this organism takes considerable time.

In the above classes of cases, then, the application of the opsonic method of diagnosis should be of value if the test is dependable. In cases where pure cultures of the offending organism are readily obtainable the method would have merely a confirmatory value.

In cryptogenetic processes a differential diagnosis has been made by the estimation of the indices of several samples of blood, obtained at intervals of 48 hours or more, to all possible infecting agents. The bacterium toward which is exhibited low or markedly variable opsonic power in several estimations has been assumed to be the important infecting agent, for purposes of vaccination.

Results of estimation of the opsonic index:

I. Staphylococcus.

Acne—19 cases. In 14 cases the initial indices were below .8; in 5 the initial indices were within normal limits. Lowest record .19.

Furunculosis—39 initial indices recorded. Of these 26 were below .8. 10 were within normal limits. 3 were above 1.2.

Other staphylococcic processes—In 53 cases the initial indices were below .8. In 10 cases the index was between .8 and 1.2. In 4 the index was above 1.2.

II. Streptococcus.

Puerperal Sepsis. 29 cases.

Eleven cases showed initial indices varying from .58 to .76. Sixteen cases showed initial indices varying from .82 to 1.13. Two cases showed initial indices of 1.29 and 1.37 respectively.

Local Processes. Five cases have shown indices varying between .4 and .8. Three cases have shown indices be-

tween .8 and 1.2. In 5 cases with infection by other organisms than the streptococcus, the streptococcus initial index was between .8 and 1.2. In 1 case with B. Coli infection (pure) the initial index was 1.40 to streptococcus.

III. Pneumococcus.

In pneumococcus infections the initial indices were .95 (pneumonia) and 1.13 (empyema).

IV. B. Coli.

In 16 cases in which B. Coli infection has been suspected or demonstrated, the initial indices have been below .8 in 12 cases, within normal limits in 4 cases, two of which were not suffering from B. Coli infection, and above 1.2 in two cases.

V. B. Pyocyaneus.

In two local infections with B. Pyocyaneus the index was below normal.

VI. Tubercle Bacillus.

1. Normals.

In normal individuals from whom a single blood has been obtained, the results have been as follows:

Nine have shown T. B. indices between .85 and 1.18.

Two normal individuals have shown T. B. indices of .70 and .76 respectively.

With two or more bloods:

In seven individuals the T. B. index varied between .80 and 1.13.

In one normal individual T. B. indices were .66 and .70.

In one normal individual T. B. indices were .62, .93 and .82.

2. Tubercular.

In most cases blood has been brought to the laboratory without any information as to the character of process. In the diagnostic application of the opsonic index to cases in which the tubercle bacillus was a possible or an

actual factor, our results are tabulated as successes or failures. Under the head of successes are included the observations in cases in which the tubercle bacillus was demonstrated to be the infecting agent, and the opsonic index lay without normal limits, *i. e.*, below .80 or above 1.2. Included here also are the cases in which the tubercle bacillus was excluded by clinical or laboratory methods, or both, as a possible infecting agent, and the index lay within normal limits, *i. e.*, between .8 and 1.2.

Under failures, have been placed all cases of tuberculosis in which the opsonic index was within normal limits.

Under doubtful, have been classified cases where two bloods have been obtained and where the indices have been contradictory, *e. g.*, where one index has been within normal limits and the second outside of normal limits. It should be said that in doubtful cases resort is always had to the examination of additional specimens of blood, where possible.

Results of opsonic estimations in local processes, other than pulmonary, where single specimens of blood were available:

Thirty-nine successes, 22 failures.

Results where more than one blood was available:

Eighty-five successes, 17 doubtful, 7 failures. Pulmonary processes (two or more bloods,) 15 successes, 2 doubtful, 2 failures.

The finding of a negative phase following the initial injection of Tuberculin T. R. has been a constant phenomenon in cases of tuberculosis studied by us.

The relative specificity of the opsonic findings has been brought out, perhaps, in the most marked way by the post operative reaction. In cases where the local tubercular focus itself has been the seat of operation, the post-operative negative phase has been much more marked than in cases where the operation has been practiced at a distance from the local focus.

Post Operative Opsonic Reaction in Bone and Joint Processes to T. B.

Tubercular-operation at site of local focus:

	Before operation.	After operation.
T. B. hip	.66	.34
" knee	.78	.30
" hip	1.02	.60

Tubercular-operation at distance from local focus:

	Before.	After.
Tubercular ankle	.83	.76
Lung T. B.*	.89	.60

Non-Tubercular.

	Before.	After.
Gonorrheal arthritis	.91	1.19
Exostosis of astragalus	.85	1.11
Exostosis of tibial tubercle	.90	1.14
Gonorrhœal arthritis	.88	.94

Control of Positive Cases. The results have been controlled in many cases by operation and the demonstration of a tubercular process in the tissues; in many cases the tubercle bacillus has been cultivated directly from the lesions or indirectly after passage through guinea pigs; in cases in which the demonstration of tubercle bacilli in smears was possible, this has been done. In a few cases dependence has been placed on clinical findings in which X-ray examinations have played an important part.

Control of Negative Cases. In some negative cases the examination of tissues obtained from the lesions has resulted in the demonstration of the non-tubercular nature of processes. The important control has been the clinical course of cases after the opsonic test had been applied.

FAILURES.

Below are listed the cases in which the opsonic findings did not conform to the pathological or clinical findings.

* Operation on osteomyelitis of tibia.

Notable in all cases, with the exception of the case of far-advanced pulmonary tuberculosis, has been the excellent general condition of the patients.

Failures. Local processes.

1. Lupus 1.01, 1.06. Wright and others have called attention to the high indices shown in this condition.

2. Tubercular Knee, diagnosticated clinically as infectious arthritis. Indices 1.00, 1.02, 1.03. Operation and later manipulation. Grossly, tissues corresponded to papillary structures found in infectious knees. Microscopical examination revealed a tubercular process.

3. Old Potts with several healed, and one discharging sinus. Indices 1.16, 1.02, 1.03, .67.

4. Tubercular abscess of chest wall. Thought to be non-tubercular. Inoculation of guinea pig resulted in development of tuberculosis. Indices .83, .81, 1.01.

5. Disease of right hip of 12 years duration. Arthrotomy. Tubercular synovia and rice bodies removed. Small cavity in inner tuberosity of tibia curetted. Indices .85, 1.08, 1.14, .95.

6. Old tubercular hip with sinuses. Indices 1.07, .95, .88.

7. Old tubercular hip with healed sinuses. Indices .96, 85.

Failures in Pulmonary Cases.

1. Apparently cured pulmonary tuberculosis. Indices taken to determine where patient stood. .92, .93, .85, .86.

2. Far advanced pulmonary tuberculosis. .94, .80, .78.

CRYPTOGENETIC PROCESSES.

Polyarthritides.

Six cases of polyarthritis in which differential opsonic tests were made, showed lower indices to the streptococcus than to other organisms.

Case A.

Strepto.	1.12	T. B.	.90	S. P. A.	.91
	.72		1.02		.97
	.70		.98		

Case B.

Strepto.	.68	T. B.	.90
	.47		1.20
	.46		.89

Case C.

Strepto.	.59	S. P. A.	.66	T. B.	.85	Pneumo.	87
	.76						

Case D.

Strepto.	.70	S. P. A.	.94	T. B.	1.12	Pneumo.	83
	.78		1.01				1.05
	.65						

Case E.

Strepto.	.75	S. P. A.	1.19	Pneumo.	1.04
	.66		.97		1.22

Case F.

Strepto.	.60	S. P. A.	.70	T. B.	.90	B. Coli.	.90

In four cases of polyarthritis, opsonic indices have been lower to the pneumococcus than to other organisms.

Case A.

Pneumo.	Strepto.	S. P. A.	T. B.
.70	.79	1.09	.84
.81	1.00	1.16	.78
.58	1.00	1.03	1.20

Case B.

Pneumo.	Strepto.	S. P. A.	T. B.
.70	.95	1.04	1.15
.79	.82	1.32	
.74		.55	

Case C.

Pneumo.	Strepto.	T. B.
.59	.72	1.13
.58	.87	1.12
.55	1.12	.92

Case D.

Pneumo.	Strepto.	S. P. A.	T. B.	B. Coli.
.44	.91	.91	.78	1.05
.33	.97		.75	
	1.04			

In one case of polyarthritis the opsonic index to T. B. was lower than that to other organisms.

T. B.	Strepto.	S. P. A.	B. Coli.
.58	.85	.90	.83
.49	.91	.86	.97

In nine cases of polyarthritis the indices to the bacterium found in Lane (see treatment) were lower than to other organisms studied.

In two cases of B. Coli subinfection (?), indices were as follows:

A.	T. B.	S. P. A.	Strepto.	B. Coli.	Pneumo.*
	1.06	.70	.92	.81	.59
	.83	.80		.62	.75
				.83	.88
				1.54	.83
B.	T. B.	S. P. A.	Strepto.	B. Coli.	Pneumo.
	.85	.60	1.00	.74	1.31
	1.12	.60	.98	.40	.67
	1.76	1.23	1.09	.60	.98
	.91				

In two cases of mucous colitis, initial indices were .69 and .52.

CASES ILLUSTRATIVE OF SUCCESSFUL DIAGNOSIS.

Positive Diagnoses.

D. I. Diagnosticated clinically as spondylitis deformans. Opsonic indices were .66, 60, .64 to T. B. Diagnosis of T. B. spine. Findings confirmed later by X-ray.

D. II. T. B. Knee, diagnosticated clinically as prepatellar bursitis. Indices .39, .50 and .32.2. Diagnosis confirmed by clinical course.

* Intercurrent cold.

D. III. Blood obtained from supposedly normal individual was found to have an index of .73 to B. Tuberculosis. Investigation showed that he had a quiescent lumbar Potts.

D. IV. Case of bronchitis, which on physical examination was pronounced doubtful as to tuberculosis. X-ray also doubtful. No T. B. found in sputum. Indices .65, .72 and .66—opsonic diagnosis of tnberculosis confirmed by finding T. B. in digested and centrifugated sputum.

D. V. Case diagnosticated as hypertrophic arthritis, with marked exostoses of acetabulum. Old tuberculin test negative. Opsonic index to T. B. .57. Operation with removal of exostoses which were found to be tubercular.

D. VI. Case diagnosticated and treated as syphilitic dactylitis without response. Indices to T. B. .45, .59. Opsonic diagnosis of tuberculosis confirmed by X-ray and results of tuberculin treatment.

D. VII. Dactylitis—doubtful clinically, though thought to be tubercular. Opsonic indices .55, .47, .48. Opsonic diagnosis confirmed by X-ray.

D. VIII. Osteomyelitis (S. P. A.) Indices to T. B. of .54, .89 and after opening of tibial abscess .60. Later clinical investigation showed a pulmonary tubercular process.

D. IX. Pulmonary process, diagnosticated clinically as probably non tubercular. Indices .85, 1.92, .52 and .70. Later examination of sputum confirmed opsonic diagnosis of active tubercular process.

D. X. A clinical diagnosis of chronic bronchitis, probably non-tubercular. Indices to T. B. .52, and .42. Diagnosis of tubercular process confirmed by clinical course.

Negative Diagnoses.

D. XI. Chronic osteomyelitis in individual, whose brother and three sisters died of tuberculosis. Opsonic indices .88, .67 (old blood), 1.22, and .91. Negative diagnosis confirmed by guinea pig inoculation of discharge.

D. XII. Chronic osteomyelitis with marked pallor. Indices to T. B. .94, .96. Negative diagnosis confirmed by clinical course.

D. XIII. Chronic osteomyelitis of 35 years standing with long intervals of quiescence. Indices to T. B. 1.14, .98, .90. Negative diagnosis confirmed by clinical course.

D. XIV. Sarcoma of knee, thought to be possibly tubercular.

Indices.	S. P. A.	Strepto.	T. B.
	.88	.87	.91
	.94	1.08	.78
			1.2

Diagnosis of non-infectious process confirmed by operation.

D. XV. Pneumonic process in child. Clinical diagnosis, probably tubercular.

Indices.	Pneumo.	Strepto.	S. P. A.	T. B.
	.48	.94	.70	1.00
	.94	.82	.92	1.20
	.69		.86	.93

Non-tubercular character of process indicated by clinical recovery in three weeks.

D. XVI. Progressive loss of weight and strength with daily rise in temperature. Tuberculosis probably excluded by repeated normal indices—.85, 1.12, .76, 1.12, .91, 1.00. Clinical course under treatment with B. Coli vaccine confirmed diagnosis of non-tubercular character of process.

D. XVII. Infection of metartarsal bones, thought to be tubercular. Indices .91, 90, .83. Operation later confirmed diagnosis of non-tubercular process.

B. Treatment.

General Considerations concerning the use of Vaccines in Treatment.

In the treatment of cases which are described in the following pages the vaccines used have been autogenous, whenever a bacterium was obtainable from the lesion, and the character of the lesion indicated that the organism which was isolated possessed a satisfactory virulence. With the Streptococcus, B. Coli and B. Pyocyaneus, an attempt has been made to obtain autogenous vaccines in every case.

With the Staphylococcus Pyogenes Aureus, a mixed vaccine made from several strains, derived from active processes, and grown for as short a time as possible on artificial media, has given best results.

Vaccines as put up in bottles covered with rubber caps, and kept in ice chest, have remained free from contamination for indefinite periods. Vaccines have been injected into the arm, usually just above elbow along outer edge of biceps, or into the back. In a few cases injection has been practised "up stream" from the point of infection, *i. e.*, in the region whose lymphatic drainage area included that of the point of infection.

Local Reaction.

The local reaction following injection of vaccines consists of slight local swelling, often associated with an erysipelatoid blush extending for a varying distance about the point of injection. The redness and swelling usually disappear in 24 to 36 hours, leaving a small, deep nodule marking the region of deposit of vaccine in tissue. This in turn rapidly disappears.

In a few individuals more marked swellings have appeared, and have required a longer time to subside. This was particularly true in the case of one individual with angio-neurotic œdema. Marked reactions are rare and are apparently a matter of idiosyncrasy.

Abscess formation has never occurred in this series of several thousand injections made by many different operators.

With Tuberculin the reaction is slight or entirely lacking.

Harmful Results of Vaccination.

Several rumors of harmful results of vaccination have come to our ears. Investigation of a report emanating from an able medical consultant, brought out the statement that in two cases of general infection in which death was immi-

nent, the fatal issue had followed shortly after the exhibition of a vaccine. That he did not personally object to the use of vaccines in such cases, however, was indicated by the fact that he agreed to the application of vaccine treatment in a case of septic endocarditis which he had seen. (Case not reported because of limited period under observation—under treatment now.) Other rumors to the effect that vaccination had been followed in some cases by general disturbances, indefinite in character, but marked by permanent lassitude and loss of working capacity, have been investigated, but specific cases in which this was true have not been found. This rumor probably had its origin in the fact that the exhibition of largedoses of S. P. A. vaccine, in individuals not sufficiently immunized is followed during the negative phase, *i. e.*, for 24 hours or less following vaccination, by a feeling of lassitude and somnolence. The reaction from this is usually sharp, and patients generally comment upon the fact that during what corresponds to the positive phase, they never felt better in their lives.

Initial doses of B. Coli in acute processes are usually followed by a depression of temperature. In extreme cases the temperature may become markedly subnormal. In one acute case the injection of 50,000,000 (a large dose in an acute case) was followed by the development of a subnormal temperature, associated with collapse, but response to warmth and stimulation was rapid.

In one case a dose of 650,000,000 B. Pyocyaneus vaccine was followed by malaise and general discomfort lasting for several days.

The repeated and long continued injection of living cultures of S. P. Aureus into animals is followed by chronic interstitial changes, notably in the liver and kidney. While it is true that the dosage of bacteria necessary to evoke these results in animals is relatively hundreds of times that used in therapeutic inoculations, it has been sug-

gested that the vaccine treatment could not but produce some changes in the kidneys of individuals who underwent treatment for long periods. A careful investigation of the urine of patients who have had for long periods injections of large doses of S. P. Aureus vaccine has shown constantly the absence of albumen at the end of treatment, where kidneys were normal at the initiation of treatment. In two cases in which albumen was present at the beginning of treatment its amount was distinctly less after treatment for months. Albuminuria appears in bacterial infections in man, either where the process is due to an actively virulent organism (acute nephritis), or where a less virulent bacterium has been active for long periods (*e. g.*, amyloid disease associated with prolonged suppuration or tubercular or syphylitic changes and, probably, chronic nephritides). That vaccine treatment may be the means of lightening the toxic burden thrown upon the kidneys is indicated in Case CLXXIX, in which the clinical evidences of amyloid disease are much less after treatment than they had been at any time since the case came under observation.

Inquiry as to the present condition of cases which underwent a course of vaccination several months ago brings again the general response: "Never felt better in my life." A number of cases report a considerable increase of weight since they underwent treatment. Whether this is due to the natural reaction following an infection, or is related to the use of vaccines, is a question.

Vaccines are used only under conditions of infection. They are injected into lymphatic areas from which the elements (endotoxins) capable of stimulating a reaction of the immunizing machinery are quickly absorbed in quantities sufficient to awaken this mechanism, which is slumbering under insufficient stimulation. It is to be presumed that, in natural infections, the absorption of these bodies is con-

stantly taking place from the focus of infection, but in insufficient quantities to elicit an immunizing response at any given time. This is especially true in local infections. Even under vaccine treatment, a certain tolerance, as yet unexplained, is developed after a time, which makes it necessary to increase the frequency of vaccination or the doses of vaccine in order to bring about the usual reaction. With Tuberculin this seems to be less true than with other bacterial vaccines. The response to small doses (1-1000 to 1-600 mg.) of Tuberculin seems to be as active after months of treatment as in the beginning. The opsonic reaction to Tuberculin is slow, the negative phase occupying usually the full 24 hour period following vaccination, and the positive phase flowing and ebbing during a period which measures about a week in most cases. In vaccination with other organisms the negative phase may be transient and the flow and ebb of the positive phase may after a time be completed in two or three days, making desirable more frequent inoculation; or the response may be absent or so slight as to call for larger doses.

Treatment of Local Processes.

Staphylococcus. Initial injections in adults 100,000,000 to 250,000,000, increased to 500,000,000 or 1,000,000,000. Maximum 1,500,000,000. In children 50,000,000 to 100,000,000. Interval 1 week to 3 or 4 days.

Streptococcus. Initial injection in adults 50,000,000, increased to 200,000,000. Interval 1 week to 3 or 4 days.

Pneumococcus. 50,000,000 to 200,000,000. Interval 1 week to 3 or 4 days.

B. Coli. Initial injections 25,000,000 to 50,000,000, increased to 100,000,000 to 200,000,000. Interval 1 week to 3 or 4 days.

B. Proteus. 100,000,000 to 200,000,000. Interval 1 week to 3 or 4 days.

B. Pyocyaneus. 100,000,000 to 250,000,000. Interval 1 week to 3 or 4 days.

Gonococcus. 25,000,000 to 200,000,000. Interval 1 week to 3 or 4 days.

T. B. Initial injection 1/2000 mg., increased to 1/1000 mg. Maximum 1/600 mg. Interval 1 week.

Treatment of General Processes.

In the treatment of general processes, other than tubercular, vaccines are used in small doses at more frequent intervals than in local processes. With the Streptococcus a dose of 12,500,000 to 25,000,000 is used. With B. Coli the amount is 25,000,000 to 50,000,000. The usual interval is at least 48 hours to permit of the development of a positive phase.

Method of Recording Cases.

Some of the cases whose results are recorded in the following pages were treated in institutions; others by various observers in private practice, and some at the laboratory. In all cases vaccine treatment was carried out *in addition* to the ordinary methods of treatment, unless otherwise indicated.

In dealing with a new methed of treatment, there is a temptation to overstate the results of treatment, especially if these results are good enough to awaken enthusiasm. In recording results, we have, therefore, tried to be exceedingly conservative and, where possible, have made use of the exact wording of comments made by clinical observers.

We have followed the example of Wright and his school in recording the highest index reached by each case. It is not to be assumed that the index rose progressively to the point recorded or that it was sustained at the high point for any considerable period. The indices of patients have been taken, as a whole, at a point corresponding to the

resting stage of the blood, and the high indices recorded by some observers, who have made estimations at a period corresponding to the apex of the curve of the positive phase, are not shown.

It is impossible, without continued vaccination, to keep the opsonic index of an individual who has been treated, high above the normal. Subsidence of the index to or below the normal usually follows after a period of varying length, on the cessation of vaccination.

Cases are classified etiologically, according to the important infecting agent.*

STAPHYLOCOCCUS INFECTIONS.

Furunculosis. Twenty-nine cases (I to XXIX) have been treated in adults. In two of these calcium sulphide had been used without result. Twenty-five cases have been cured and discharged. Of these, five cases recurred, but responded to one injection of S. P. A. vaccine. One recurrence is shown in chart. Two cases were under treatment irregularly and final results are not known. Two cases under treatment too short a time for conclusions. One injection of stock vaccine usually gave good results where the process was limited and in an early stage. Initial indices were, as a rule, low, varying from .38 to .85. Cases under treatment have shown indices as high as 1.79. Cases which have required more than one or two injections have had a mixed autogenous and stock vaccine.

Illustrative cases. I. Male, 33. Follicular infection of anterior nares more or less constant for last year. Carbuncle

*We are indebted for the use of records and clinical material, and for other courtesies, to members of the visiting and house staffs of the Boston City Hospital, the Carney Hospital, the Boston Dispensary, the Mass. Eye and Ear Infirmary, U. S. Naval Hospital, U. S. Marine Hospital, the House of the Good Samaritan, St. Elizabeth's Hospital, St. Mary's Infant Asylum, and the Cushing Hospital, as well as to many physicians in Boston and eastern Massachusetts, to whom we here extend our thanks.

on neck and lip during this time. March 25, under observation with marked pustule in right nares and on the lip. Index to S. P. A. .71. Received 5 injections to 9th of May varying from 350 to 650 million S. P. A. vaccine. Discharged May 9, with index of 1.10. Cured. As a matter of prophylaxis was told to report in 8 weeks; on July 2 index was .75. 650 mil. S. P. A. given. Has remained free from furuncles since.

II. Three years ago had a felon, destroying distal phalanx of finger. For last year, troubled with boils. Came under observation April 1, with a large furuncule on neck. S. P. A. index .55. Vaccine injected on the 3d and in three days discharge had stopped. Following a second injection on the 9th, a large angry boil appeared but never pointed, gradually disappearing. Six injections varying from 250 to 500 million S. P. A. Discharged May 16. Aug. 2, patient appeared again with large boil on forearm. Cultures showed pure S. P. A. Aug. 3, was given 450 million S. P. A. vaccine. Boil cleared up. Immunity apparently lasted 2½ months.

Furunculosis in children. Twelve cases (XXX to XLI) were treated in one institution. In this institution the development of furunculosis has usually been associated with other evidences of lowered resistance, and has progressed to a fatal issue, with symptoms of a general septic process in more than a majority of cases. Under vaccine treatment, "all cases have shown improvement." Three cases died of associated condition immediately after the initiation of treatment. Nine cases progressed favorably to healing of lesions accompanied by a marked improvement of general condition and are cured. Four or five injections of S. P. A. vaccine in doses of 50,000,000 over a period of 3 to 4 weeks have been necessary. Two cases (XLII and XLIII) have been treated for a limited period in another institution with only slow reaction and less marked results. Treatment discontinued.

Carbuncles.

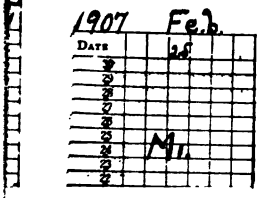
Five cases have been treated. Two carbuncles of back of neck, (XLIV and XLV) treated without operation. Duration of process 15 and 21 days. Healing was progressive, with absence of pain and development of slight scars, a single dimple in each case. Indices .80 and 1.03, went to 1.07 and 1.33 respectively.

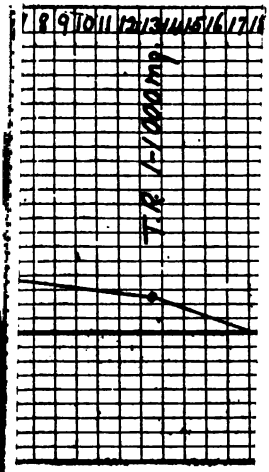
XLVI. One carbuncle of the back of the neck which had been incised, and was practically healed, developed a ring of pustule about wound. These disappeared within 24 hours after one injection of 250,000,000 S.P.A. Later several furuncles developed on neck. Treatment irregular. After complete healing there was a recurrence. Present condition unknown. Initial index .84, went to 1.03 under vaccination. One carbuncle of neck (XLVII) associated with furunculosis—required treatment for one month before all processes cleared up. Initial index .38, went to 1.02 under treatment. No recurrence.

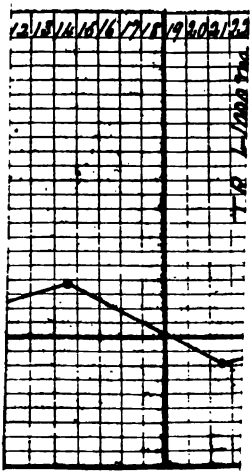
XLVIII. Carbuncle of lip—duration 5 months. Under treatment with X-rays and ultra violet rays—has recurred with constancy. Infection of mucosa of soft palate by tongue. (Pure culture S. P. A. from pustules on edge of ulcer which occupied larger part of upper lip.) Under vaccination showed improvement for 48 hours, following large doses of S. P. A. vaccine. After this period activity of process would be reawakened. On vaccination every 2d day with 1,000,000,000 S. P. A., processes in palate and lip rapidly healed. Since this a recurrence in two places on lip has occurred, following traumatism by dentist, but is healed.

Septic hands. Three cases.

XLIX. In one a process involving first phalanx of initial finger. Incision and corrosive dressing was followed by extension of process and swelling of whole hand. An injection of S. P. A. vaccine, 500,000,000, was followed by immediate relief—the swelling wholly disappeared within 3 days and incisions healed rapidly. Indices .75, .65, .96. (Culture S. P. A.)


1907 Feb.
Date
Mi.


8 9 10 11 12 13 14 15 16 17 18
T.B. 1-1000 atm.


12 13 14 15 16 17 18 19 20 21 22
T.B. 1/1000 atm.

T. B. CURVE.

XLIX A and B. In the other two cases a single vaccination was made following incision. Subsidence and healing rapid. (Culture S. P. A. Initial indices .82, .76 respectively.)

L. *Septic finger*—following tearing out of finger by Hamburger steak machine. Stump gangrenous—lymphatic extension along arm. A standard dose (250,000,000) S. P. A. vaccine was given under a misunderstanding at the time when lymphatic extension and general intoxication were most marked. Index 1.29. This was followed by marked depression and malaise lasting for about 24 hours. The next day, however, the general condition was markedly improved—the stump was clean, and with a second injection 3 days later process rapidly healed.

Osteomyelitis—7 cases treated.

LI. Osteomyelitis of femur (culture S. P. A.) followed typhoid 35 years ago, recurring 9 years ago and 1 year ago is cured. Index from .8 to 1.52, last index 1.26.

LII. Osteomyelitis of femur following operation for ununited fracture 1 year ago is still under treatment (3 months). Initial indices .99 and .81, has gone to 2.78. Last index 2.49. (Culture S. P. A.)

LIII. Osteomyelitis of tibia, fibula and femur with purulent synovitis of knee joint, following compound fracture one month ago. Initial index .73, has gone to 1.69. Last index 1.53. The case when coming under treatment was toxic, with evening temperature of 100 to 102; pulse 100 to 120. The question of amputation was seriously considered. Cultures showed S. P. Aureus and B. Pyocyaneus. Under treatment with Aureus and Pyocyaneus vaccines, temperature became normal in one week. General condition rapidly improved. Pyocyaneus injections stopped when this organism ceased to be cultivable. S. P. Aureus vaccine continued. Several sinuses persisted, but discharge grew progressively less. Recently, operation for sequestra, followed by closure of sinuses.

LIV. Osteomyelitis—multiple processes following typhoid. Culture shows S. P. A., and many contaminating organisms. Not improved under vaccinations, though still under treatment. Initial indices, 1.14, .92, went to 1.4 under treatment.

LV. Osteomyelitis in tibia of 9 years' duration, with swelling of leg (associated pulmonary tuberculosis). Amputation repeatedly advised. After index, which had been .7, was raised to 1.25, operation, consisting of removal of sequestrum occupying whole length of bone, and filllng gutter with iodoform jelly, was practiced. Healing had been progressive under vaccination. (Pure culture S. P. A.) Leg now being used with cane. Tuberculosis T. R. for lung process with disappearance of all clinical signs (see tubercular processes).

LVI. Osteomyelitis of humerus. Operation removal of sequestrum and use of iodoform jelly—vaccination to control mild reinfection—successful.

LVI A. Osteomyelitis of tarsal bones—operation with resection of 1st and 2d metatarso-phalangeal joints. Initial index .4 has gone to 1.78 under vaccination. Process is quiescent at times, and has healed once, but after healing reopened and small sinus persists after two months.

Eczema—5 cases. LVII to LXI.

1—cured. See chart.
2—improved.
1—scalp—recurred.
1—eloped.

Initial indices. .33, .6, .65, .67, and .61, respectively. Last indices 1.2, 1.29, .40 (disappeared). .61—1.41 (scalp). (All showed S. P. Aureus in culture).

Illustrative case.

LVII. Facial eczema since birth. A child 6 months old. Culture from exuded serum shows pure S. P. Aureus. Initial indices .33 and .49. Injections began 4/29, .07. 4/30, redness gone—well in patches. An injection of 50 million 5/3, was followed by a violent inflammatory reaction, the eczema being much worse. Intercurrent digestive disturbance. 5/8 very suddenly the eczematous areas have cleared up, the skin is peeling in patches all over face, leaving a clean smooth surface—whole process over by the evening of 5/9. No itching. Digestive upset also passed off and child practically free from eczema until 5/15, when a few small patches appeared on cheeks. (Index 5/13 .67).

5/15, injection was followed by improvement next day but not so marked as formerly, a few patches remaining on cheeks. Has been well since 5/21. (See chart.)

The correspondence between the low index the 13th and recurrence of the eczema on the 15th is notable. Vaccination on the 14th was recommended on account of the low index, but the case looked so well that the recommendation was not carried out.

Acne—19 cases treated. (LXII to LXXX.)

4 indurated.
10 pustular.
5 pustular and indurated.

Initial indices have varied from point .19 to .96. Most of the cases have shown indices from .5 to .7.

1 case (pustular) shows complete cure. 7 cases (1 pustular—1 indurated) show almost complete cure—a rare pustule appearing at long intervals.

4 cases show marked improvement—occasional recurrences at short intervals.

4 cases discontinued treatment.

3 cases showed no improvement after treatment for one month.

Illustrative cases.

LXII. Acne at intervals for last eight years. Three months ago crop of pustules appeared on forehead. Observation Mar. 6—S. P. A. index .75. Treated to May 10 with injections of S. P. A. varying from 150 to 700 mil. Discharged cured. June 20, last report, no recurrence.

LXIII. S. Acne indurata ten years' standing. X-ray with failure—much improved under S. P. A. vaccination.

LXIV. M. Pustular acne of face for 2 years. Treatment with S. P. A. vaccine since April 19. Improvement. At present face clears up completely soon after an injection but after an interval of 14 days without vaccination pimples again appear. Still under treatment.

LXV. B. A marked pustular acne which had persisted in spite of treatment for 10 years, except for a short period following an attack of erysipelas. Initial indices 1.10, .99.

Under treatment, three months—index went to 1.4. Patient has returned twice with single pustules. Otherwise cure practically complete.

LXVI. An extreme type of pustular acne. Initial indices 1.09, .82—went to 1.21 under treatment, which was irregular. Process practically cleaned up except for few pustules. Discontinued treatment against advice, with recurrence.

LXVII. Case of acne indurata — initial index .68. Went to 1.3 under treatment with disappearance of process except for small patch of quiescent nodules which persisted for some weeks—face now clear.

LXVIII. Pustular acne of back—13 years' duration. Treated by washes and X-ray for 5 years. Showed more marked improvement under X-rays than under other treatment. Treatment with vaccines for 3 months—resulted in clearing up process to a degree never attained before—no pustules for several weeks—few small indurated nodules. No new processes for one week.

LXIX. Acne indurata and pustula for 2 years. Under treatment with washes for 2 months without result—under vaccine treatment for six weeks—process completely cleaned up, but since shows a rare indurated nodule. During latter part of treatment received 1,500,000 S. P. Aureus at weekly intervals. Indices .61, .61 went to .90 under treatment.

LXX. Acne indurata and pustula of face—5 years duration. Initial index .58—has gone to 1.4 under treatment. Under treatment 2 months—shows only slight improvement.

LXXI. Pustular acne of body—initial index 1.06—has gone to 1.39 under treatment. Process cleared up in 2½ months.

LXXII to LXXX, inclusive, were under treatment too short a time for conclusions.

In all cases cultures showed S. P. Albus with an Aureus which was a slow pigment producer (1 week). Autogenous vaccines were used in three cases without good results in the beginning of the work. Stock vaccines made from several strains of virulent S. P. Aureus have been used

since. Doses have varied from 250,000,000 to 1,000,000,000, once or twice a week.

Sycosis—3 cases.

LXXXI. Pustular Sycosis of face—under treatment by skin specialists for 5 years without relief. Under vaccination, process has cleared up except for rare pustule.

LXXXII. Sycosis Barbæ—universal—large pustular type—under medical treatment several months without avail. Under vaccination, process early cleared up except for focus under chin which was resistant. Vaccinations every 2 days with slowly progressive disappearance of all foci. Cured.

LXXXIII. Sycosis Barbæ—Eight years duration with recurrences. Under treatment one month. Initial indices .48 and .36. Went to 1.08 under treatment—distinct improvement.

LXXXIV. *Hordeolum*—One case in which 7 sties had followed progressively along right upper and lower lids, resisting treatment. Cured by a single vaccination. (Culture S. P. A.)

LXXXV. *Impetigo*—One case of impetigo contagiosa—face and neck. Resistant to treatment. Crusts wiped off leaving a clear skin in three days following a single vaccination.

LXXXVI. *Purulent rhinitis*—One case, refractory to general treatment and recurrent at frequent intervals. Process so severe that nose had to be freed mechanically. Initial index .48—went to 1.65 under treatment. Last index 1.39—Cured. (Culture S. P. A.)

LXXXVII. *Chronic rhinitis*—in which S. P. A. predominated in cultures. Is still under treatment. Amelioration of symptoms only temporary. Indices 84, .60, .95.

LXXXVIII. *Empyema of antrum*—One case cured by two injections (culture S. P. A.). Initial indices .62 and .68—went to 1.28 under treatment.

LXXXIX. *Breast abscess*—one case. Breast abcesses occurring during lactation. Refractory to surgical procedures. Under vaccination proceeded rapidly to cure. Initial index .75, went to 1.95 under treatment.

XC. *Parotitis*—One case cured following two injections. Initial index .74, went to 1.23 under treatment. (Culture S. P. A.)

XCI. *Septic burn*—one case. Suppuration following hot water burn. Child of 10. No pus, 48° after single dose S. P. A. vaccine. Rapid healing—culture S. P. A.

XCII. *Frontal sinusitis* of 4 years' duration with sinus. Vaccination followed by diminution of discharge which is now a slight serous ooze. Culture S. P. A.

XCIII. *Double otitis media with mastoiditis*—case which seemed to be proceeding toward a mastoid operation showed considerable improvement (disappearance of acute symptoms) under vaccination. Initial indices .72 and .60 —went to 1.02 under vaccination. (Culture S. P. A.)

XCIV., XCV. Two cases of *unilateral mastoid*, evidently becoming operative, were cleared up under single vaccinations.

CONCLUSION.

The results of vaccination in staphylococcic processes have been so constantly good that comment is unnecessary.

STREPTOCOCCUS INFECTIONS.

a—Uterine Sepsis.

Procedure. In cases of uterine sepsis an attempt has been made to supply an autogenous vaccine within 24 hours of receiving cultures. An early experience taught the necessity of care in obtaining cultures. A culture taken, by a competent specialist, from the uterus of a case of post-puerperal sepsis, showed a pure growth of B. Coli. Colon vaccine was made and injected. It was followed by some improvement in temperature and pulse, but the reaction was only temporary. A persistent low opsonic index to the Streptococcus suggested that this organism was working in association with B. Coli. Five numbered swabs were passed into the uterine canal to the fundus and were then planted on blood serum. Swab 1 gave rise to a pure culture of B. Coli. Culture from Swab 2 showed few streptococci in the

water of condensation, but growth was principally made up of colonies of B. Coli. With cultures from the other swabs there was progressive increase of Streptococcus and decrease of B. Coli colonies. Pure cultures of Streptococcus were readily obtainable in direct transplants from tube 5.

During a recent service in the gynecological wards of one institution it has been the custom to make cultures from the uterine canal of all cases of uterine sepsis either on admission or during curetting. From these cultures vaccines have been prepared, when the growth permitted and the clinical indications, (persistent fever) suggested a possible need of vaccination.

In 26 cases cultures have been taken. In 7 cases no growth occurred. Four of these cases showed no temperature and were presumably non-bacterial. One showed very slight temperature for several days. Two died later of puerpueral sepsis. In these cases it is possible that the alcohol or corrosive solution used in treatment prevented growth.

In 19 cases, culture showed growth. In 15 of these a streptococcus was the predominating organism. In 2 it was in close association with B. Coli. In one, growth showed a mixture of B. Coli and S. P. Aureus. In one an almost pure growth of a Pneumococcus was obtained.

Vaccines were made in 15 cases and were used in 12 cases of this series.

Nineteen cases of streptococcic uterine sepsis (XCVI to CIV) from various sources have been treated. Of these 17 were puerperal and 2 followed miscarriage. In all cases vaccination was practiced in addition to treatment by the ordinary methods of surgical procedure. In two cases vaccines were exhibited 3 hours and 20 hours, respectively, before death, without result.

In 12 cases with fever, the exhibition of an *autogenous* vaccine was followed by a drop of temperature to the nor-

mal. In nine of these cases drop was immediate—in one preceded by a slight rise. In three cases the lowering of temperature was slower, taking 2, 3 and 5 days respectively, to reach normal. In three cases a slight recrudescence responded immediately to a second dose of vaccine.

In one case, one, and in a second case two injections of 10 strain stock vaccine were followed by disappearance of fever and subsequent normal temperature.

In one case the injection of a stock (1 strain) vaccine was followed in 48 hours by continuous normal temperature.

In one case associated with pus tubes, vaccination was followed by a slow return of temperature to normal.

In three cases vaccines were used after temperature had reached normal and there was no secondary rise.

b—Other Pelvic Processes.

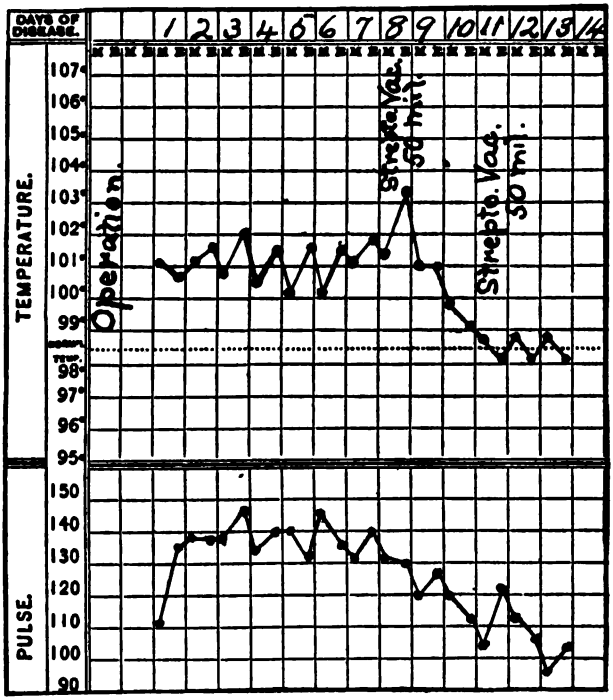
CV. One case of *pelvic abscess*, after miscarriage at three months, received one injection on 6th day when temperature was normal. Small sinus left on discharge. Progress satisfactory.

CVI. One case, *extra-uterine*, followed by sepsis. One injection on 5th day resulted in normal temperature on 6th. No subsequent rise. Sinus closed rapidly.

CVII. One case of *vaginal section* following puerperal sepsis, with a temperature from 100 to 102.8. A vaccine was made, but through an error was not given. 50 million streptococci (autogenous) given on 8th day after operation when temperature was 102.8. This injection was followed by a slight rise in temperature, a drop on the next morning to 101, and a progressive drop to normal in three days. (See chart on opposite page).

The series reported above is too small for conclusions, but the constancy with which the lowering of temperature followed autogenous vaccination (especially in cases where the drop in temperature was unrelated to operation) and the absence of marked recrudescence of the process are at least suggestive. A point which has impressed the medical

men in touch with most of the cases has been the constancy with which vaccinated cases have left the hospital with "uterus of normal size, in normal position, and freely movable."



OPSONIC SERUM.

In very acute cases of puerperal sepsis with rapid general infection the attempt to establish an active immunity by the use of vaccines has usually met with failure. A response to stimuli which demand active efforts on the part of the tissues cannot be furnished by such patients at the time when vaccination is usually called for, *i. e.*, as a last resort. To meet this difficulty we are endeavoring to immunize rabbits to many strains of streptococci by the use of killed

cultures, subcutaneously and later intravenously. In emergencies, blood can be obtained by cardiac puncture, the serum separated and injected into the patient within an hour or two of its removal from the rabbit. Such a serum, while locally toxic (*i. e.*, at the point of inoculation) should contain all of the elements which have been produced in response to the injection. The deterioration of so-called bacteriolytic sera, under the conditions which ordinarily obtain before it is delivered to the physician, is probably in part responsible for the unsatisfactory results following their use. The commercial anti-sera are prepared by subcutaneous inoculations alone, and Besredka and others have found this method unsatisfactory, the introduction of the cultures intravenously being necessary if a high degree of immunity response was desired.

It is interesting to note that the first intravenous injection of a many strain streptococcus vaccine into rabbits robbed the serum of these animals *immediately* of almost all opsonic power. The opsonic response to vaccination has been slow but definite.

Streptococcus opsonic serum has been so far used only in one case, in which injection was practised so shortly before death that no results were to be expected.

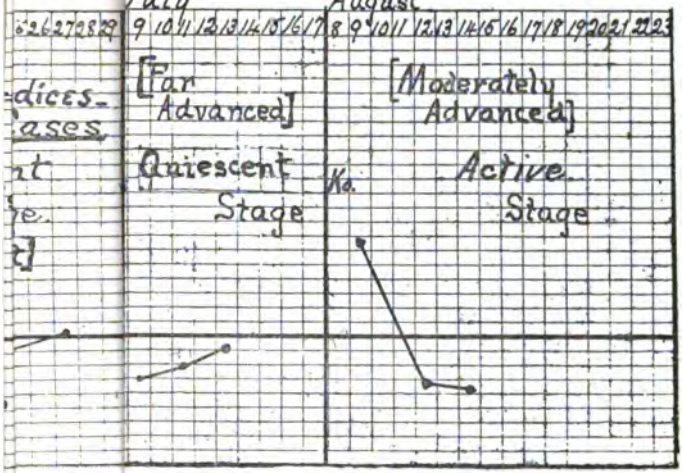
c—Other Streptococcic Infections.

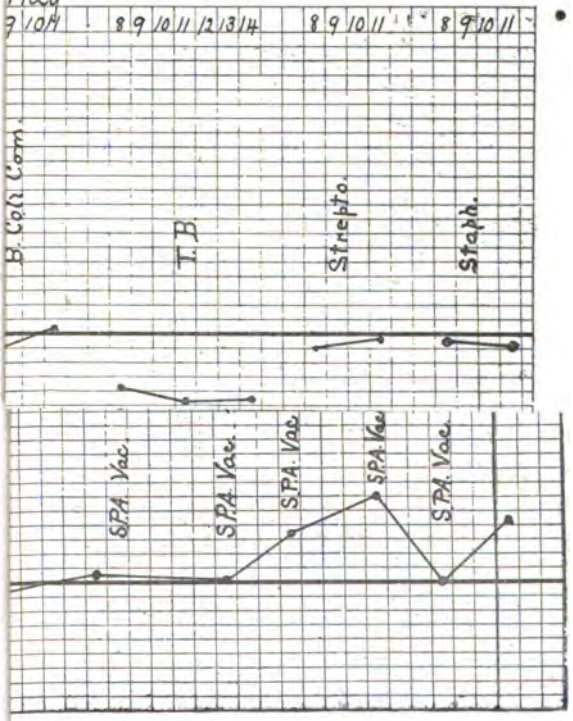
CVIII. One case of *traumatic osteomyelytis* of jaw (3 months, bilateral, with sinuses) improved steadily under vaccination—sinuses closing after two months (culture streptococcus and S. P. Aureus).

CIX. One case of *erysipelas*—improvement slow under vaccination (culture streptococcus).

CX. One *septic finger* in which amputation was seriously considered on account of extension, improved rapidly following vaccination (cultures streptococcus).

CXI. Acute *cervical abscess* which had persisted for 6 weeks was cured following 2 vaccinations. Initial indices .82, .79 went to 1.24 under vaccination (culture streptococcus).


July
August
[Far Advanced]
Quiescent Stage
[Moderately Advanced]
Active Stage


May
B. Coli Com.
T.B.
Strepto.
Staph.
S.P.A. Vac.
S.P.A. Vac.
S.P.A. Vac.
S.P.A. Vac.
S.P.A. Vac.

CXII and CXIII. Two cases of acute streptococcic *tonsillitis* were followed by return of temperature to normal and relief of symptoms within 24 hours after single injection of 10 strain stock vaccine (25,000,000) (cultures Streptococcus).

CXIV. One case *empyema—not treated with vaccines* but interesting because watched opsonically, following operation. Initial index .54, going progressively to 1.24 in three weeks, accompanying cure. Autogenous vaccine prepared, but not used, since clinical and opsonic progress was satisfactory.

CXV. One case *empyema*, with sinus, of two years' duration. Drainage of cavity unsatisfactory. Culture showed Streptococcus—later Streptococcus and S. P. Aureus. Vaccination with autogenous Streptococcus vaccine resulted in a temporary diminution of discharge, but after a time lower part of cavity filled up and there was an acute febrile disturbance. This cycle repeated several times. Under proper drainage and suction treatment, following a second operation, streptococci disappeared from pus, being replaced by S. P. Aureus, which vaccines (S. P. A. stock) caused to disappear. Vaccine treatment in this case has been distinctly unsatisfactory. Sinus is almost healed under proper surgical measures.

Polyarthritis.

In six cases of polyarthritis the blood serum, examined opsonically, was low to the Streptococcus and high to other organisms. The results of treatment with stock Streptococcus vaccines are indicated below:

CXVI. 1. Dec. 17, 1906. For past six days complains of chills, pains through body, and vomiting. Right hand became swollen and painful. Left hand, feet, ankles, knees and shoulders affected. Put on rheumatic treatment. Friction in chest—pains in joints called for morphia. Gradual improvement to Jan. 9. Left elbow became very bad —required morphia every night. Knee much worse. Lies with leg in extreme flexion. Opsonic index low to Streptococcus, .68, .45 .45. Went to 1.75 under treatment. Three injections of Streptococcus vaccine, beginning Feb.

12, followed by great relief from pain. Patient able to get about with crutches.

CXVII. 2. Polyarthritis began with slight attack in elbow in 1901. Later right knee, right ankle, back, neck, left wrist, left shoulder, jaws, right wrist, left knee and left ankle were affected in the order mentioned. Treatment here and abroad for several years without result. Streptococcus and Pneumococcus indices low. Treatment with Streptococcus and Pneumococcus vaccines with relief of pain at times. Streptococcus index went from .75 to 1.4 under treatment. Pneumococcus index from .43 to 1.4 under treatment.

CXVIII. 3.—Left ankle, left knee, right wrist and hand, and left wrist affected in the order mentioned. Process in ankle persisted. Streptococcus indices .78, .50 went to 1.88 under treatment. Five injections of Streptococcus vaccine—35,000,000 to 75,000,000. Under vaccination and baking, pain was relieved, motion of joint increased, and patient became able to bear weight on foot.

CXIX. 4.—Process in knee joints of several weeks' duration, following acute tonsilitis. Under vaccination and baking, process became quiescent in two months with partial restoration of function and relief of pain. Later, however, patient had an attack of acute pericarditis, which kept him in bed for several months. During this time his joints became entirely normal. Initial indices .70 and .78 went to 1.26 under treatment.

CXX. 5.—Acute process of both knees. Under vaccination and baking, symptoms subsided with cure. Initial index .62 went to 1.68 under treatment.

CXXI. 6.—Acute process of knees and ankles, under vaccination without other treatment. Pain immediately relieved and process rapidly subsided after two vaccinations. Initial indices .60 and .75.

CXXII. In one case of *Arthritis Deformans* of 4 years' duration, feet, ankles with bursæ, shoulders and fingers of both hands were affected. Patient unable to walk. After vaccination for two months, is free from pain except in hands, and is able to walk. General condition much improved. Streptococcic index has gone from .59 to 1.74 under treatment.

CXXIII. One case Still's disease—with persistent low Streptococcus indices, was treated with Streptococcus vaccine without results. Initial indices .68 .58 .62—went to 1.65 under vaccination.

This series of joint cases is too small to justify conclusions. Relief of pain has been an almost constant result of vaccination. Whether other good results are to be related to the vaccine, the other therapeutic measures used, or to the well-known intermittent character of the disease must remain a question.

PNEUMOCOCCUS INFECTIONS.

Our observations of infections in which the Pneumococcus was the principle infecting agent, have been limited to two cases of pneumonia, and one old case of empyema. In several cases of polyarthritis, the index has been lower to the Pneumococcus than to other organisms.

CXXIV. One case of *pneumonia*, treated with Pneumococcus vaccine progressed favorably. The second case was not treated with a vaccine.

In the cases of polyarthritis the results of treatment have corresponded to the results obtained from the use of Streptococcus vaccines in cases whose indices to the Streptococcus were low (*q. v.*).

BACILLUS COLI INFECTIONS.

Acute Conditions.

CXXV. *Pelvic abscess*, which had been incised and drained. Patient entered hospital with temperature of 102°, pulse 130. Mass filling left side of pelvis and extending almost to umbilicus. After operation (dilatation of sinus and drainage), patient, a particularly intelligent woman, became dull, semi-comatose, with vomiting of everything ingested. Cultures from pelvic abscess showed pure B. Coli. A stock vaccine was exhibited in dose of 50,000,000. Within 24 hours of injection, patient's mental activity was restored, and within 48 hours regular diet was tolerated without disturbance. Vaccination with au-

togenous vaccine, repeated at intervals of 4 to 7 days, was accompanied by a gradual drop of temperature to normal, associated with progressive shrinkage of mass in left side. This, at discharge, was in size less than that of a small orange. Since discharge, mass has again given rise to slight pain accompanied by an increase of leucorrhœa, which was controlled by two vaccinations of 50,000,000 each.

CXXVI. B. Coli. *Peritonitis*—laparotomy—multiple omental abscesses (B. Coli). Later, breaking down of tissues about abdominal wound—fecal fistula—recrudescence of process with rise of temperature, and general evidences of a toxic disturbance. Vaccination (50,000,000 autogenous B. Coli) followed by development of sub-normal temperature with collapse, which, under stimulation, was rapidly recovered from. Under two injections of vaccine recovery progressive—fecal fistula healed in two weeks. B. Coli indices, initial .50—went to 1.15 under vaccination.

CXXVII. *Gangrenous appendicitis with general peritonitis*—operation. B. Coli in pure culture in turbid peritoneal fluid. Autogenous vaccine injected 24 hours after operation, followed by rapid drop of temperature, and repeated vaccinations by cure. Initial indices .79, .53—went to .94 under vaccination.

CXXVIII. *Peri-appendiceal abscess.* Operation. B. Coli and S. P. Aureus in culture. Mixed vaccines followed by rapid healing of sinus and cure. Initial index B. Coli .51—S. P. A. .79. B. Coli index went to 1.31 under vaccination.

CXXIX. *Fecal fistula* on the 5th day after operation for pyosalpynx—temperature 100.5. A single injection of 50,000,000 B. Coli vaccine was given, followed by drop to normal next day. Fecal fistula closed 4 days after injection. Sinus persisted without vaccination for some time. On the re-application of vaccines, sinus closed after 6 vaccinations at 2 and 3 day intervals. (Culture—B. Coli.)

CXXX. *Appendix Abscess.* Temperature 100 to 101 24 hours after operation. Fecal fistula developed on next

day, when wick was pulled. 50,000,000 B. Coli vaccine given. Temperature dropped to normal the following day, and fecal fistula closed in 3 days. (Culture—B. Coli.)

Chronic Conditions.

CXXXI. *Intestinal Tuberculosis*—had 12-15 movements daily, at times with large amounts of blood. Had lost 40 lbs. Appendicostomy with lavage of colon and Tuberculin. No diarrhœa and no blood for three weeks following operation. On getting out of bed, blood began to reappear in stools. Was put to bed for a time and vaccination with autogenous B. Coli vaccines practised. Under combined vaccines with daily colonic lavage (.85% NaCl) has gained progressively. Is up and about. Weight has returned to normal—general condition excellent—feels practically well. A recurrence of diarrhœa, (4 daily movements) with streaks of blood, was controlled by increasing B. Coli vaccine from 100 to 160 million. Vaccines weekly. Tuberculin T. R. (Tuesday), and B. Coli (Friday), except during reawakening of diarrhœa, when B. Coli vaccine replaced Tuberculin on Tuesday. (See chart.)

CXXXII. In a second case of *Tubercular Enteritis* accompanied by diarrhœic movements, containing large quantities of purulent material, the amount of pus is controlled by the injection of B. Coli vaccines. Withdrawal of vaccine is followed constantly by increase of pus, which shows B. Coli in almost pure culture. The general condition is progressing, however, although patient at last report showed slight improvement.

Cystitis.

Six cases have been treated.

CXXXIII.—*Cystitis*, *ureteritis* and *pyelo-nephritis* with dysuria for 3 years, showed on examination left ureter size of thumb. Retroverted uterus containing 2 small myomata. Operation—D. & C.—myomectomy—invagination of appendix—suspensio uteri. At operation, left ureter, starting 4' from bladder to brim of pelvis, was thickened to size of thumb, and about midway, a small soft nodule was felt. Two weeks after operation, urine showed large quantities of pus—ureteral condition persisted. A

pig inoculated with sediment did not give tubercular reaction. Culture showed B. Coli. An autogenous vaccine was injected in three doses of 50,000,000 at intervals of 5 and 7 days. Pus progressively decreased as did B. Coli. Patient discharged with a clear urine, containing slightest possible trace of albumen, a rare granular cast, no pus and no bacteria. No bladder symptoms. "Ureter no larger than clay pipe stem on leaving."

CXXXIV. *Catheter cystitis* of one year duration. Frequent micturition. Nocturia 4 times. General condition poor. Urine contains large quantities of pus, from which B. Coli and a poorly pigmented S. P. Aureus were obtained on culture. Treatment for 4 months with autogenous B. Coli vaccine followed by marked general improvement, freedom from nocturia and abatement of frequency during daytime. Urine still shows free pus and bacteria. Initial index .55 went to 1.81.

CXXXV. *Prostate cystitis* of three years' duration. Poor general condition, nocturia. Urine showed alkaline fermentation with abundant pus. Cultures showed B. Coli. Treatment with autogenous B. Coli vaccine for 5 months, accompanied by abatement of nocturia and marked improvement of general condition. Much mucus, few bacteria and little pus. Initial index to B. Coli, 1.19, has gone to 4.58 under treatment.

CXXXVI. *Cystitis* and *pyelitis* of 20 years duration. General condition poor. Has considerable pain and nocturia. Sediment shows free pus, and occasional blood cells. Culture of urine shows an abundant growth of B. Coli, with a few colonies S. P. Albus. G. pig negative to T. B. on inoculation. Initial index to colon .84. Treatment with autogenous vaccine (B. Coli) in doses of 50 to 150 million once a week. General condition is somewhat improved—local condition better at times. B. Coli still abundant on culture. Pain and frequency lessened.

CXXXVII. A marked case of *cystitis* in a child, of 6 months' duration. General condition poor, micturition frequent. Culture showed B. Coli in pure culture. Initial indices .75 and .72. Has gone to 1.52 under treatment. Treatment for one month, with autogenous vaccine, in doses varying from 30 to 75 million, together with local

treatment, resulted in improvement of general condition. The patient has been too short a time under treatment for final conclusions.

CXXXIX. *Cystitis.* A case of 40 years duration showed little improvement under vaccination. Initial indices .67, .69—went to 2.03 under treatment. Only 3 injections given. Patient compelled to go away.

B. Coli Subinfection(?).

The following two cases were treated with B. Coli vaccines, solely because in differential diagnosis, based upon the opsonic index, the indices to B. Coli were constantly low. Treatment in each case has been followed by amelioration of symptoms. In the first case, the constipation from which the patient suffered has been completely relieved. In the second case the temperature and loss of weight have been corrected.

CXL. Influenza 15 years ago. Shifting pains in body since. Left middle ear 10 years ago. Some trouble with right ear at same time. Since has not felt strong, but had no real illness until 1 year ago when she had an attack of rheumatism. In bed one week. At present does not feel strong. Complains of aches and pains with changes in weather. Bowels inactive—must take cathartic each night. Initial indices to B. Coli .81, .62. Rapidly improved, under vaccination. Constipation cured. General condition much improved. Appetite good.

CXLI. Pelvic operation several years ago followed by fecal fistula which finally healed. Patient's general condition has been poor since. For last three years has been growing gradually worse. Running a temperature of 100–101 and losing weight. Various methods of treatment resorted to with failure. Differential diagnosis opsonically showed B. Coli indices constantly low.

3/18	.74
3/20	.40
3/26	.60

Treatment with stock colon vaccine which was obtained from an appendicitis-peritonitis case, also with an S. P. A.

stock vaccine, according to opsonic indications. Doses of B. Coli vaccine varied from 35 to 125 million—of S. P. A. vaccine, from 50 to 60 million. After three months' treatment there was a marked improvement of general condition and temperature was normal.

No conclusions are of course justified from these two cases. The results are, however, tangible enough to suggest the application of this method of treatment to a large series of cases. The possible relation of intestinal intoxication or subinfection to intestinal or other neuroses, either as a cause of the process, or as a result of the lowered resistance which accompanies it, is a subject which has received much attention and which will bear further study along these lines. At the time of writing we are coming in touch with some cases of membranous entertitis and mucous colitis with lowered B. Coli indices, which we intend to follow from this standpoint.

General Conclusions—B. Coli Infections.

The results of treatment of acute B. Coli infections have been uniformly satisfactory. In chronic processes results have not been so definite in all cases. A notable disappointment has been the persistence of bacteriuria in chronic urinary infections, although other urinary symptoms have been relieved.

B. PYOCYANEUS INFECTIONS.

In addition to several cases in which this organism was found as a secondary infecting agent, it occurred as the predominating organism in one case (CXLII) in which there was a fecal fistula and *peri-appendical abscess*, following operation upon a gangrenous appendix in which a pin was found. Patient was toxic, with continued slight temperature and progressive loss of weight and strength. Cultures showed B. Pyocyaneus and B. Lactis Aerogenes. Exhibition of B. Pyocyaneus vaccine in doses of 200,000,000 was followed by immediate improvement and accompanied by

rapid closing of fistula and healing of sinus. B. Pyocyaneus index went from .60 to 1.41 under treatment.

In cases where B. Pyocyaneus was present in association with other organisms, it disappeared quickly after the exhibition of Pyocyaneus vaccine.

B. PROTEUS INFECTIONS.

Four cases of *cystitis* in which proteus-like organisms predominated have been under treatment. In two of these cases tuberculosis of the genito-urinary tract has been present. In one case (CXLIII) the associated use of Proteus vaccine together with Tuberculin has been followed by relief of nocturia and clearing up of the urine. In this case the urine has cleared up relatively at times under other treatment, but has never remained clear for as long a period as under present treatment.

In a second case (CXLIV) of bacteriuria of 12 years' duration, the injection of proteus vaccine has been followed constantly by a discharge of large quantities of mucus, shreds and pus, persisting for 10 or 12 hours, to be followed by the passage of a relatively clear urine. The bacteriuria still persists, however, although less marked than in the beginning of treatment. The third and fourth cases were under treatment for too short a time for definite conclusions.

The results of the vaccine treatment in this class of cases has been on the whole disappointing. Frequent micturition, nocturia and pain have been relieved, but bacteriuria has persisted.

DIPLOCOCCUS INTRACELLULARIS MENINGITIS.

CXLVI. Child, 5 years. A case of *cerebro-spinal meningitis* came under observation just before death. Fluid from lumbar puncture showed meningococcocci in smear and culture. A single dose of autogenous vaccine was given within a few hours of death. Meningococcus index 24 hours before death 1.03—from post mortem blood 1.53.

INFECTIONS WITH B. TUBERCULOSIS.

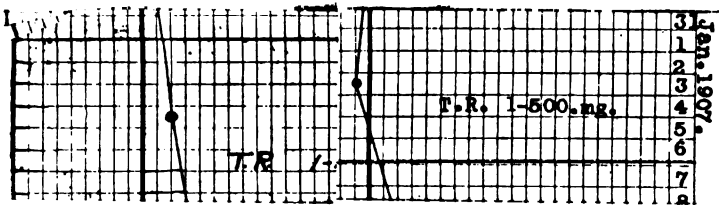
The treatment of tuberculosis with various tuberculins has been practised for many years. The tuberculin which best satisfies requirements from the vaccine standpoint, is Koch's Tuberculin T. R. This is a watery suspension of dried powdered tubercle bacilli, produced mechanically by trituration. The Wright method of procedure overcomes the objections which have been raised to its use according to the method recommended by Koch.

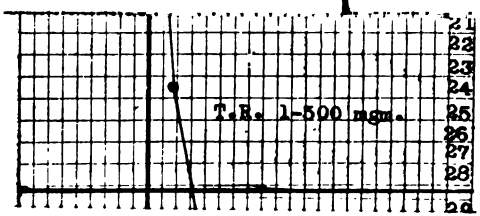
In early preparations of T. R. Tuberculin, which is a mechanical suspension of the crushed bodies of tubercle bacilli, living tubercle bacilli were found. A reasonable objection to the use of material which might contain living bacteria has been met by the sterilization of the tuberculin, and the use of a carbolized solution for its dilution (see technique).

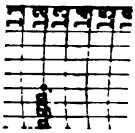
Too large or too frequent doses of Tuberculin T. R. will be followed by marked clinical reaction, and evidences of intoxication. Wright found that minimal doses would bring about a satisfactory opsonic response (rise in the opsonic index) and that the rise in the index would persist for a week or more. He therefore recommended standard doses of 1/1000 mg. dried powder, with 1/2000 mg. as an initial dose, and 1/600 mg. as the maximum dose. The injection of proper doses will be followed by absolutely no general clinical reaction. If a clinical reaction follows vaccination, it may be concluded that an overdose has been given.

Pulmonary Tuberculosis.

The following cases of pulmonary tuberculosis have been under treatment with Tuberculin T. R. in addition to such hygienic treatment as could be carried out in patients' homes. None of the cases were treated in sanatoria.


T.R. 1-500.mg.
T.R. 1-
Jan. 1907.
31
1
2
3
4
5
6
7


T.R. 1-500 mgm.
21
22
23
24
25
26
27
28



CXLVII. Incipient pulmonary tuberculosis. Physical signs very slight. T. B. present in sputum. The initial indices were .64, .79, .72, and under treatment went to 1.45. Tuberculin T. R. was given in doses of 1–1000 to 1–500 mg. weekly for four months. Physical signs disappeared. Case apparently cured.

CXLVIII—Incipient pulmonary tuberculosis, in quiescent stage, of one year's duration. Some pigmentation of skin and mucosa of mouth present. There is a question of Addison's disease in this case. The initial indices were .56, .62, .59, and went to 1.54 under treatment. The case has been treated with Tuberculin T. R. for six months. The pigmentation has disappeared, the patient is much stronger and has gained in weight. Still under treatment.

CXLIX. Pulmonary tuberculosis, far advanced, and unfavorable for treatment. Process is pretty extensive throughout one lung. T. B. are present in sputum. The initial indices were .94, .80, 78, went to 1.40. Treatment with Tuberculin T. R. has been followed out for 8 months. The case has been progressive, the physical signs remaining about the same. For a time he gained in weight but has since dropped back again. Case is still under treatment.

CL. Phthisis—far advanced. Initial indices .65, .74, .88. Went to 1.61. Disease progressing—no improvement—under treatment 2 months.

CLI. Incipient pulmonary tuberculosis. Under treatment 5 months. Indices have gone from .64 to 1.42. In good condition.

CLII. Incipient pulmonary tuberculosis—good general condition. Indices .79, .66, have gone as high as 1.22 under vaccination. Present condition excellent.

LIII. Incipient Pulmonary tuberculosis associated with S. P. A. osteomyeletis of tibia. All lung signs have disappeared—present condition good.

CLIII. Pulmonary Tuberculosis with a tubercular bursitis of elbow of 4 months duration. Initial index .73, went to 1.59 under treatment. Case was given injections of Tuberculin T. R. for four months but has not been under treatment since July. Nothing can be said as to result in this case.

Tuberbulosis of Lymph Node.

CLVI. *Tabes Mesenterica.*? Previous history shows that patient had an attack of pleurisy in 1904, at which time T. B. were found in sputum. About one year later patient had a throbbing pain in back, which was followed by a swelling over right buttock. Swelling was incised and considerable pus was evacuated. The sinus was traced to 4th lumbar vertebra but no bare bone was found. Wound healed but shortly broke out again with a free discharge of pus. At this time he came under observation. His initial index was .56 to T. B. Treatment with Tuberculin T. R. was given weekly for 5 months. At the end of this time treatment was discontinued. Since treatment has been stopped he is reported to have pulmonary tuberculosis.

CLVII. *Tubercular cervical glands.* Has suffered with attacks of tonsillitis for last 12 years. Since a bad attack three years ago cervical glands have gradually enlarged to size of pullet's egg. Right side larger than left. Mar. 1, 1907, tonsils removed and found to be tubercular. Mar. 8, 1907, all superficial glands and some deep glands on right side of neck removed and drainage inserted. Came under observation March 21. The swelling of right side was still marked and sinus was discharging. Culture showed S. P. Aureus. The initial indices to S. P. A. were .82 and 1.15, and to T. B. .40 and .66. S. P. A. index went to 1.49 and T. B. to 1.25 under treatment. Treatment with S. P. Aureus and Tuberculin T. R. began April 25. The swelling gradually subsided and discharge stopped. Slight induration around scar only remains. No glands to be felt.

CLVIII. Child—5 years. A superficial *cervical gland* size of pigeon's egg which has been present one year. Infection followed fall down stairs with a trumpet in mouth. Mouthpiece was driven deeply into left tonsil, which showed deep ulceration and, after healing, left a deep depression in region of tonsil. Under treatment 3 months. Gland remains quiescent. Condition of child, although good at first, is better now. Indices have gone from .47 to 1.4 and have been constantly high since beginning of treatment.

CLX. *Tubercular sinus of neck* of 2 months' dura-

tion. Initial index to T. B. .53, .82, .88—going to 1.42. Treated with weekly injections of Tnberculin T. R. in doses ranging from 1–1000 to 1–250 mg. Has been under treatment for 5 months and is slowly improving.

CLXI. *Tubercular glands in axilla* of 1 month duration. The initial indices were .45, .72, .83, and went to .98. Has been treated for two months with Tuberculin T. R. in doses from 1/1000 to 1/750 mg. General condition improved, glands quiescent.

CLXII. *Tubercular cervical glands* of five years' duration. Four years ago glands on left side of neck suppurated, an incision was made and drainage instituted. Sinus discharged for some time, but is now healed. Glands on right side of neck enlarged about 1 year ago, to size of walnut. At this time the initial T. B. indices were .72, .74, .86. Has been treated for 8 months with Tuberculin T. R. in doses of 1/1000 to 1–750 mg. Indices have gone to 1.40. General condition better—glands show no change.

CLXIII. *Tubercular cervical glands* of 2 months' duration. Initial indices to T. B. were 1.21—.68, and went to 1.72 under treatment. After 6 injections of 1/500 to 1/250 mg. (overdose) at intervals of seven days, the index dropped from 1.18 to .82. Case has been under treatment for 5 months and shows slight improvement in general condition. Glands quiescent.

Tubercular Empyema and Caries of Rib.

CLIV. Empyema followed by caries of rib and sinus. Duration 1½ years. The initial indices to T. B. were .69, .61, .56. Under treatment went to 1.74. S. P. Aureus was .60, 53, and went to 1.56. Has been treated with Tuberculin T. R. and S. P. Aureus vaccine since April 6. At the beginning there was a marked diminution in amount of discharge, which improvement did not last. A culture on August 5 showed streptococci and the index to Streptococcus was .76. Under autogenous Streptococcus vaccine the index went to 1.21, and the discharge has markedly diminished. Treatment discontinued on account of absence.

CLV. Empyema followed by caries of rib and sinus, of 1½ yrs. duration. Culture showed S. P. Aureus. The initial indices to T. B. were .53, .85, 1.31, and under treat-

ment went to 1.52. S. P. Aureus indices went from .62, .73, .74, to 1.73 under treatment. Treatment with Tuberculin T. R. and S. P. A. vaccine began on June 7. July 17 a culture showed Streptococcus to be the predominating organism and on the 19th autogenous Streptococcus vaccine was given. The index on the 19th was .80 and under treatment went to 1.35. After the exhibition of Streptococcus vaccine the discharge was very much lessened. At present a slight discharge still persists.

Lupus.

CLXIV. Lupus of face of 30 years' duration with destruction of nasal cartilages and closure of right nostril following plastic operation. Has been treated with X-rays, violet rays, caustics, and excision. When seen, showed an irregular butterfly ulceration including whole of nose and upper lip and extending widely into cheeks beyond malar eminences. Initial indices 1.01, 1.06. Under treatment 5 months. Tuberculin T. R. 1/1000 mg. twice per week for 5 months. Ulceration gradually healed. Face wholly clear within two months, but since an occasional small ulcer has appeared. Face has been wholly clear of ulcers for past month. Scars are superficial. Here and there shot-like nodules can be felt. Still under treatment.

Genito-urinary Tuberculosis.

One case (see CXLIII) has been under treatment for two months.

CLXV. Patient had a tubercular hip when a child. In January, 1907, tubercular kidney was removed. In May, 1907, began to have pain if urine was held longer than two hours, and also after urinating. At intervals passed blood at the end of micturition. Has to get up 5 to 10 times at night to urinate. Initial indices to T. B. were .60, .85, .55. Went to 1.58. Patient was treated for 5 weeks with Tuberculin T. R. in doses from 1/1500 to 1/750 mg. Patient then went away for the summer. Pain and frequency of urination somewhat lessened.

Intestinal Tuberculosis.

Two cases (see CXXXI and CXXXII) have been under treatment for 4 and 7 months, respectively. In one the

progress of the disease is arrested and patient is in excellent condition. In the other case, treatment has been irregular and progress is slow.

Tuberculosis of Bones and Joints.

CLXVI. Tubercular *hip* with discharging sinuses. Initial indices of .60, .48, .60. Went to 1.72 under treatment. Operation, incision and removal of sequestra. Cultures of discharge showed B. Pyocyaneus and S. P. Aureus. Case was treated with Pyocyaneus and S. P. A. vaccines and Tuberculin T. R. weekly, for one month. Treatment then discontinued. Slight discharge still present.

CLXVII. Tubercular *spine* with discharging sinus. Cultures showed S. P. Aureus. Initial indices of .69, .47 to T. B. Went to 1.40 under treatment. Treated with Tuberculin T. R. and stock S. P. A. vaccine. Discharge continued about the same until autogenous S. P. A. vaccine was given. After a short time under the autogenous vaccine, discharge was very much diminished. Is still under treatment.

CLXVIII. Tubercular *spine* of ten years' duration. Cold abscess with sinuses. Drainage imperfect at times. Cultures show S. P. Aureus in pure culture. The initial T. B. indices of .76, .52—went to 1.33 under treatment. Case has been treated with S. P. A. vaccine and Tuberculin T. R. weekly, for three months. General condition improved at first but has dropped back during the last month. Discharge has continued about the same as before treatment. Still under treatment.

CLXIX. Acute tubercular *hip* of four months' duration. The initial indices were .73, 1.02. The index of 1.02 was taken just before operation. The index after the operation was .60. Under treatment index went to 1.44. After excision of hip there was an abundant purulent discharge from wound. B. Pyocyaneus found on culture. Was treated with an autogenous Pyocyaneus vaccine. After a few injections the blue color of pus disappeared and, at time he was lost sight of, discharge had diminished to 1/5 its previous amount. Weekly injections of Tuberculin T. R. were given for three months, in doses of 1/000 to 1/500 mg. General condition somewhat better.

CLXXI. *Tubercular dactylitis*, affecting middle and index fingers of left hand—duration four months. Case was treated 6 weeks with anti-syphilitic treatment without result. The initial indices to T. B. were .45, .59 and under treatment have gone to 1.50. Has been under treatment with Tuberculin T. R. in doses from 1/1000 to 1/500 mg. for five months. The patient's general condition has been much improved and the swelling of the finger has gone down to quite a noticeable extent. He can now wear a ring which could not be fitted over finger before vaccination began.

The following cases of bone and joint tuberculosis were treated in an institution where a successful effort is made to place cases under the best hygienic conditions, notably as to sunlight and fresh air.

CLXXII. Tubercular *hip*. The initial indices to T. B. were .72, .54. Under treatment index went to 1.80. Case was treated with Tuberculin T. R. for six months up to April 12. In February, hip was incised and drainage inserted. Operation was followed by irregularly high temperature. Case grew gradually worse, developing meningeal symptoms in April, and after several convulsions died.

CLXXIII. Tubercular *spine* of seven months' duration. Acute knuckle present and case had been treated by Bradford frame for 1 week, with immediate relief, when it came under observation. Initial T. B. indices of 1.47, 1.19, went to 1.54 under treatment. Treated with Tuberculin T. R. for 5 months, when treatment was discontinued. At this time patient was gaining steadily.

CLXXIV. Acute tubercular *hip*, began in August, 1906. In the next 5 months patient was operated on three times in order to establish drainage and remove sequestra. In Jan. 1906, clinical signs of beginning amyloid disease of kidney, liver and spleen became apparent. G. pig positive to T. B. Urine showed $\frac{1}{4}\%$ albumen, casts, blood and pus. Patient was running a temperature of about 100°. In Sept., abscess of groin was incised. At this time spleen was to be felt 2 fingers' breadths below costal margin. In October, a large abcess which extended over whole of sacrum was incised. At this time patient came under ob-

servation. The initial indices to T. B. were .72, .88. Under treatment the index went to 1.34. Case was treated with Tuberculin T. R. in addition to ordinary measures for five months. Urine in Dec. showed large trace of albumen and many casts. While under treatment general condition improved. The last note on March 7, 1907, reports patient to be in fair condition.

CLXXV. Patient came under observation Aug. 18, 1906, with right *hip* ankylosed and with acute signs in left hip. All previous sinuses were healed. The T. B. index on Aug. 31 was .64. Treatment with Tuberculin T. R. from Oct. 4 to date. General condition good at present. Ankylosis of left hip.

CLXXVI. Tubercular *knee* of three years' duration came under observation in Sept. 1906, with anklylosis of right knee. Several discharging sinuses on knee and scars of several which have healed. There is subluxation of tibia. T. B. initial index .60 and went to 1.45. Cultures showed S. P. Aureus. Treated with Tuberculin T. R. and S. P. A. vaccine. Patient has grown quite stout and the discharge diminished. Treatment stopped in Mar., 1907. General condition good. Sinuses about the same.

CLXXVII. Patient came under observation in October, 1906, with double tubercular *hips*. Several sinuses and abscess present in left groin. Abscess incised and drained. Culture showed streptococcus. The T. B. initial indices of .58, .88 went to 1.30. Initial indices to Streptococcus were .72 and .84 and went to 1.50 under vaccination. Treated with Tuberculin T. R. 1/1000–1/500 and autogenous Streptococcus vaccine in doses of 250 million. The discharge from sinuses cleared up in 2 months and sinuses healed. Slight amount of motion in each hip. General condition very good. Discharged March 9, 1907.

CLXXVIII. Chronic tubercular *spine* with tubercular *hip*. Case had just been operated upon when it came under observation. At that time there were three sinuses which extended to the pelvis, evidently coming from psoas region. From Sept. 1 to Oct. 16, sinuses were exposed daily to direct rays of the sun. The discharge became less and general condition improved. Early in October opsonic

initial indices were .35, .73, 75. Case was treated with Tuberculin T. R. for 10 months. In addition Streptococcus and S. P. A. vaccines were also injected, but for shorter periods. The T. B. index went to 1.46 under treatment. In Jan. general condition was much improved. During March, 1907, patient had slight set back, and jacket was removed. No tenderness or spasm of back was discovered, however, and in May, patient was able to be out of doors. Has considerable cough. There are some coarse dry rales to be heard in lungs. Improvement marked during summer. Patient is able to walk without crutches and is free from pain. General condition is excellent. Still under treatment.

CLXXIX. Tubercular *knees* with discharging sinuses. Right *hip* also tubercular, with fluctuating swelling over joint. Incontinence of urine and feces. Emaciated. Large amyloid liver, spleen, and kidneys. Liver on a line with umbilicus. The discharge from the sinuses had become somewhat less when the case came under treatment with Tuberculin T. R. in December. The initial indices to T. B. were .46, .73, .77, .61 and went to 1.45. During Jan., 1907, the general condition remained about the same. Later regained control of sphincters, but passed urine into bed. Under education the bed-wetting ceased. In February the fluctuating mass over right hip was incised and drained.

By May the general condition of patient was much improved. Sinuses were discharging very little. The liver had become much smaller and patient was up in go-cart. During July the sinuses on both legs were practically healed. Liver was then about 2 fingers' breadths below costal margin. Patient had gained much in weight and is bright and active.

At present case is wearing light plaster casts on legs and is trying to walk with crutches. Sinuses are entirely healed. General condition is excellent. Has a few degrees of permanent flexion and a few degrees of free motion in each knee. Liver is now not over one finger-breadth below costal margin. This case has been treated with Tuberculin T. R. for about 9 months in doses from 1/000 to 1/500 mg. Cultures of discharges showed S. P. Aureus

and S. P. A. vaccine was also given during a part of the time.

GENERAL CONCLUSIONS.—TUBERCULAR INFECTIONS.

None of our cases have been under treatment for a sufficient length of time to justify final conclusions.

Results as a whole have been encouraging enough, however, to warrant the opinion that Tuberculin T. R., in minimal doses according to the Wright procedure, is a valuable therapeutic agent. In no cases has it done harm. In several cases results have been obtained which are better than could have been expected from other methods of treatment alone.

CASES UNDER TREATMENT FOR SHORT PERIOD.

We have not considered it necessary to record in detail a large series of cases which were under treatment for too short a time to permit of results.

Thirteen cases of *tuberculosis of bone and joints* received from one to nine injections of Tuberculin T. R. The cases which received eight injections or more are here detailed:

One case received eight injections. *Acute tubercular hip.* Test with old tuberculin negative. Initial index .57. Operation. Microscopical examination of exostoses showed abundant miliary tubercles. Treatment was followed by temporary slight improvement, but was discontinued. Three months later patient was admitted to hospital, where he died of phthisis.

One case received nine injections. Potts with paraplegia. Initial index .83—went to 1.40 under treatment. Improvement very slight.

Eight cases of *pulmonary tuberculosis* have received from one to ten injections of Tuberculin, T. R.

The following cases received eight injections or more:

Incipient pulmonary tuberculosis—patient is a recent graduate of a sanatorium with quiescent process. Initial indices 1.36, 1.33, .83. Eight injections Tuberculin T. R. Process quiescent—no change.

Pulmonary Tuberculosis and infectious arthritis. Initial indices .69, .70. Ten injections Tuberculin, T. R. Index went to 1.30. Process at a standstill.

Seven cases of *local tuberculosis* received from one to five injections of Tuberculin T. R. without improvement.

The following cases of *polyarthritis* which gave low initial indices to a bacterium isolated from a case of infectious polyarthritis by Dr. Rhoades Fayerweather in 1905, and known as the Lane organism, were treated with vaccines made from cultures of this organism.

30. Initial index .65. Eight injections of Lane vaccine. Index went to 1.55. Temporary improvement. Treatment discontinued.

31. Initial index .50. Went to 1.15 under vaccination. Treatment continued at patient's home with temporary improvements.

32. Initial indices 1.26, .68. Two injections Lane vaccine. Treatment discontinued.

33. Initial indices .94, .68. Index went to 2.13 under vaccination with *temporary* improvement. Treatment discontinued. This case also received several injections of Streptococcus vaccine—with indices from .66 to 1.09.

Four cases of *polyarthritis* received two to four injections of Streptococcus vaccine, without improvement. Two cases of polyarthritis received two and four injections respectively of Pneumococcus vaccine, without improvement.

Three cases of *otitis media chronica* received one or two injections of S. P. A. vaccine without improvement. One case of staphylococcic *bacteriuria* received three injections of autogenous vaccine with slight temporary improvement.

CONCLUSIONS.

1. The phagocytosis of most bacteria is possible only after the bacteria have been prepared by serum substances which Wright has called opsonins; the opsonins of normal serum are capable of preparing non-virulent bacteria or those possessing a slight degree of virulence for phagocytosis; markedly virulent organisms are prepared only by the action of the opsonins of specific anti-sera.

2. The opsonins of normal serum possess a relative degree of specificity.

3. The opsonins of immune serum (immune opsonins) are almost absolutely specific.

4. The resistance of virulent bacteria to phagocytosis is due to bodies corresponding in bio-chemical character to endotoxin.

5. Immune opsonins unite with the endotoxins of virulent bacteria much as known antitoxins unite with their specific toxins. A reasonable conclusion is that immune opsonins are therefore anti-endotoxins.

6. In the diagnosis of doubtful or cryptogenetic infectious processes, including tuberculosis, the determination of the opsonic index furnishes a valuable addendum to present methods.

7. The occurrence of an opsonic negative phase following injection of Tuberculin T. R. is a more exact diagnostic test for tuberculosis than any which we possess at present.

8. In treatment as an adjuvant to standard methods, the use of vaccines, guided by the index, where possible, has almost uniformly made the progress of cases more satisfactory and rapid than ordinary methods of treatment could have done; in many cases vaccine treatment has been followed by results which could not have been obtained by any other method of treatment.

We are deeply indebted for special clinical assistance to Dr. Benjamin E. Wood; for many opsonic estimations to Dr. Grace Rochford; for assistance and suggestions to Drs. Arthur W. Tucker and Lawrence W. Strong, as well as to Drs. Evangeline W. Young, Jennie Segal, John D. Clark and Olga Cushing Leary.

BIBLIOGRAPHY.

1. Aaron: New York Med. Journ., vol. lxxxiv, Dec. 1, 1906.
2. Baldwin: Med. News, Sept. 30, 1905.
3. Barrat: Proc. Roy. Soc., 1905, vol. lxxiv, p. 524.
4. Bergey: Journ. A.M.A., vol. xlvi, 1906, Nr. 8, p. 580.
5. Besredka: Etudes sur le bacille typhique et le bacille de la peste. Ann. de l'Inst. Past., vol. xix, 1905, p. 478.
6. Besredka: De l'anti-endotoxine typhique et des anti-endotoxines, en general. Ann. de l'Inst. Pasteur. 1906, p. 149.
7. Besredka: Des Endotoxines solubles typhique, pesteuse, et dysent'erique. Ann. de l'Inst. Pasteur. 1906, p. 304.
8. Bordet: Ann. de l'Inst. Pasteur., vol. ix, 1895, p. 462.
9. Bordet: Ann. de l'Inst. Pasteur., vol. xi, 1897, p. 177.
10. Bradley: Physician and Surgeon, Detroit, Dec. 1906.
11. Bradshaw: Lancet, 1906, vol. T. Nr. 20, p. 1387.
12. Bradshaw and Glynn: Liverpool Med. and Chir. Journ., May, 1906.
13. Brooke: Dublin Journ. Med. Sci., Sept. 1906.
14. Bullock: Proc. Roy. Soc., vol. lxxv.
15. Bullock: Medico. Chir. Soc. Proc., 1905.
16. Bullock: Practitioner, 1905, Nov.
17. Bullock: Trans. Path. Soc., London, 1905. Vol. lvi, pp. 334-344.
18. Bullock: The Lancet, 1905, vol. ii, p. 1603.
19. Bullock and Atkin: Proc. Roy. Soc., 1905, vol. xxiv, p. 379.
20. Bullock and Ledingham: Lancet, Dec. 21, 1905, p. 1603.
21. Bullock and Western: Proc. Roy. Soc. Series B, vol. lxxvii, p. 531.
22. Bunch: Lancet, March, 1907.
23. Bunch: Lancet, Jan. 19, 1907.
24. Calvert: Lancet, Feb. 2, 1907.
25. Calvert: Brit. Med. Jour., 1906, Nr. 2375, pp. 19-21.
26. Cheyne. Lancet, 1906. Vol. I, No. 2, pp. 78-82.
27. Clark. Jour. A.M.A., Dec. 29, 1906, p. 2159.
28. Cler. Centralb. t. Bakt. Abt. I. Orig. Bd. xxxx, 1905.
29. Craw. Jour. of Hyg., Jan. 1905, vol. v, p. 126.
30. Dean. Proc. Royal Soc., S. B., 1905. Vol. lxxvi, p. 506, also Centbl., f. Bakt., Part I, 1905. Ref. vol. xxxvii, p. 349, u. 449.

31. Denys. Resultats obtenus par le serum anti-streptococcique. Internat. Med. Congress at Moscow, 1897. Section iii, p. 82.

32. Denys and Leclef. Centbl. f. Bakt., 1898, vol. xxiv, p. 685.

33. Denys and Leclef. Sur le mecanisme de l'immunité chez le lapin vacciné contre le streptocoque pyogene. La Cellule. Vol. xi, 1895, p. 177.

34. Dorset. Am. Medicine. Vol. iii, 1902, p. 135.

35. Dodds. Brit. Med. Jour., 1906. Nr. 2375, pp. 23-24.

36. Dreyer. Brit. Med. Journ., Sept. 10, 1904.

37. Dunhill. Intercolonial Med. Journ. of Australia, Nov. 1906.

38. Ellett. Brit. Med. Journ., July 21, 1906.

39. Famulener. Central States Med. Monitor, Jan. 1907.

40. Flexner. Jour. Med. Research, 1902, vol. iii, p. 316.

41. Frazer. Glasgow Med. Jour., March, 1907.

42. French. Brit. Med. Jour., Feb. 2, 1907.

43. Grieg. Proc. Linn. Soc., N. S. Wales, 1905, pt. 2, July 26.

44. Gruber. Centralbl. f. Bakteriol. Part i. Ref. vol. xxxviii, 1906.

45. Gruber and Futaki. Munch. med. Wochenschrift, 1906. Nr. 6, p. 249.

46. Hamilton and Horton. Jour. Infect. Dis., 1906, vol. i, p. 128.

47. Hektoen. The Journal A.M.A., May 12, 1906, p. 1407.

48. Hektoen. The Journal of Infect. Dis., vol. iii. Nr. 1, March, 1906, pp. 102-109.

49. Hektoen. Transact. of the Chicago Path. Soc., vol. vi, 1906. Nr. 11, pp. 408-409.

50. Hektoen. The Journal of Infect. Dis., vol. iii. Nr. 3, May, 1906, pp. 434-440.

51. Hektoen. Jour. Infect. Dis., vol. i, 1906, p. 102.

52. Hektoen and Reudiger. Jour. Infect. Dis., 1905, vol. ii, pp. 128-141.

53. Himmel. Ann. de l'Inst. Pasteur, 1901, vol. xv, p. 928.

54. Hollister. Surgery, Gynæcology and Obstetrics, Chicago, vol. iii, No. 6, p. 740.

55. Horton (Miss). Trans. Chicago Path. Soc., 1905, vol. vi, p. 297.

56. Horton (Miss). Jour. Infect. Dis., vol. i, 1906, p. 110.

57. Houston. Brit. Med. Jour., 1905, vol. i, p. 854.

58. Houghton. Therapeutic Gazette, Detroit, Jan., 1907.

59. Huggard and Moreland. Lancet, June 3, 1905, p. 1493.

60. Issaef. Ann. de l'Inst. Pasteur., 1893, vol. vii, p. 260.

61. Kellogg. Northwest Medicine. Jan. 1907.

62. Kinghorn and Twitchell. Amer. Jour. Med. Sci., Aug., 1906, p. 203.

63. Knorr. Jour. A.M.A., April 13, 1907, p. 1255.

64. Landsteiner and Reich. Centbl. f. Bakt., Abt. 1, 1905. Orig. Bd. xxxix, p. 712.

65. Lawson and Stewart. Royal Med. and Surg. Soc., London, 1905, (Nov. 14.)

66. Lawson and Stewart. Lancet, 1905, vol. ii, pp. 1679-1684.

67. Leishmann. Note on method of quantitatively estimating the phagocytic power of the leucocytes of the blood. Brit. Med. Jour., 1902, vol. i, (11 Jan.) p. 73.

67A. Leishmann. Jour. Hyg., 1905, vol. v, p. 380.

68. Leishmann. Some experiments in connection with "stimulins." Taansact. Path. Soc., London, vol. lvi, 1905, pp. 344-355.

69. Levaditi, C. Ann. de l'Inst. Pasteur, vol. xv, 1901, p. 894.

70. Little (Graham). Brit. Jour. of Dermat., 1904, Sept.

71. Löhlein. Ann. de l'Inst. Pasteur, 1905, vol. xix, p. 647.

72. Löhlein. Ibid. Vol. xx, 1906, pp. 939-961.

73. Löhlein. Centralbl. f. Bakteriol. Abt. T, Ref., vol. xxxviii, 1905, pp. 32-36.

74. Lyon. Lancet. June 24, 1905, p. 1718.

75. Macdonald. Practitioner, Nov., 1905.

76. Mallory. Jour. Exp. Med., 1898, vol. iii, p. 611 and 1900, vol. v, p. 1.

77. Marchand. Arch. de Med. Exp., 1898, vol. x, p. 253.

78. Marino. Ann. de l'Inst. Pasteur, 1904, vol. xviii, p. 761.

79. McFarland. Medicine. April, 1906, p. 247.

80. Meakin and Wheeler. Brit. Med. Jour., Nov. 25, 1905, p. 1396, vol. ii.

81. Mennes. Zeitschrf. Hyg. u. Infectionskr. 1897, vol. xxv, p. 413.

82. Metchnikoff. Uber eine Sprosspilzkrankheit der Daphnien. Virchows Arch., 1884, vol. xcvi, p. 177.

83. Metchnikoff. Immunité des lapins, vaccinés contre hog cholera. Ann. de l'Inst. Pasteur, 1892, p. 289.

84. Metchnikoff. Recherches sur l'influence de l'organisme sur les toxines. iii memoire. Ann. de l'Inst. Pasteur, 1898, p. 263.

85. Metchnikoff. Sur la peste bubonique. Comptes rendus du xii congres International de Medicine. Moscow, 1900.

86. Metchnikoff. Lecons sur la pathologie comparée de l'Inflammation. Paris, 1902.

87. Metchnikoff. Immunitat bei Infektionskrankheiten. Deutsche Ubersetzung, June, 1902.

88. Metchnikoff. Reaction phagocytaire. Conference d'Amsterdam, 1904.

89. Miller. Therapeutic Gazette, Detroit, March, 1907.

90. Mumford. Boston Med. and Surg. Jour., March 28, 1907.

91. Muir and Martin. On the combining properties of the opsonins of immune serum. Proc. Roy. Soc. Vol. 79B, p. 187.

92. Muir and Browning. Jour. of Hyg. 1906, vol. vi, p. 20.

93. Muir and Martin. Brit. Med. Jour., Dec. 22, 1906.

94. Neisser and Wechsberg. Zeitschr. f. Hyg. u. Infektionskr., 1902, vol. xxxvi, p. 299.

95. Neufeld and Rimpau. Deutsch Med. Wochft. 1904, vol. xxx, p. 1458; also

96. Zeitft. f. Hyg. u. Infekt. 1905, vol. i, p. 283.

97. Neufeld and Topfer. Centbl. f. Bakt. Abt. I, 1905, Orig., vol. xxxviii, p. 456.

98. Ohlmacher. Jour. A.M.A., Feb. 16, 1907, p. 571.

99. Park and Williams. Jour. Exp. Med., 1905, vol. vii, p. 403.

100. Pottenger. Jour. A.M.A., April 13, 1907.

101. Portis. Jour. Infect. Dis., 1901, vol. i, p. 127.

102. Potter. Ditman and Bradley, Jour. A.M.A., Nov. 24, Dec. 1, 1906.

103. Potter. Ditman and Bradley, Amer. Jour. Med. Sci., Aug., 1906, pp. 186-202.

104. Ricketts. Trans. Chicago Path. Soc., 1903, vol. v, p. 173.

105. Rosenow. Human pneumococcic opsonin and the anti-opsonic substance in virulent pneumococci. Jour. Infect. Dis., June 15, 1907.

106. Ross. Canadian Lancet, January, 1907.

107. Ross. Brit. Med. Jour., London, July 6, 1906.

108. Ross. Brit. Med. Jour., London, Nov. 24, 1906, p. 1452.

109. Reudiger. The Journal A.M.A., 1906, vol. xlvi, p. 108.

110. Reudiger. The Journal A.M.A., 1905, vol. xliv, p. 198.

111. Reudiger. Transact. of section on Pathol. and Physiol. of Amer. Med. Ass., 1904, p. 397.

112. Reudiger. Jour. Infect. Dis., vol. iii, 1906, pp. 102-109.

113. Savtchenko and Melkich. Ann. de l'Inst. Pasteur, 1901, vol. xv, p. 498.

114. Savtchenko. Ibid., 1902, vol. xvi, p. 106.

115. Simon. Johns Hopkins Hosp. Rep., Jan., 1906.

116. Simon. Jour. A.M.A., Jan. 12, 1907, p. 139.

117. Simon. Lamar, Bispham, Jour. Exper. Med., Dec., 1906.

118. Smith. R. Greig, Proc. Linn. Soc., N. S. Wales, 1905, Part II, July 26.

119. Urwick. Brit. Med. Jour., 1905, July 22, p. 172.

120. Van de Velde. Ann. de l'Inst. Pasteur, 1896, vol. x, p. 580.

121. Walker. Jour. Med. Research, 1905, vol. xiv. Nr. i, p. 173.

122. Warthin. Trans. Chicago Path. Soc., 1903, vol. v, p. 151.

123. Weinstein. Berliner klin. Wochenschr. 1906, Nr. 30, pp. 1007-1011.

124. Western. Brit. Jour. of Dermat., August, 1905.

125. Wolf. Jour. of Infect. Dis., October, 1906.

126. Wright. Notes on the treatment of furunculosis, sycosis, and acne by the inoculation of a staphylococcus vaccine; and generally on the treatment of localized bacterial invasions by therapeutic inoculations of the corresponding bacterial vaccines. Lancet. March 29, 1902, pp. 874-884.

127. Wright. On the measurement of bactericidal power of small samples of blood under ærobic and anærobic conditions, and on the comparative bactericidal effect of human blood drawn off and tested under these contrasted conditions. Proc. Roy. Soc., vol. lxxi, 1902.

128. Wright. On some new procedures for the examination of the blood and of bacterial cultures. Under this, (2) on a method of determining under the microscope the number of micro-organisms contained in a bacterial culture. Lancet, July 5, 1902, p. 11.

129. Wright. A lecture on therapeutic inoculation of bacterial vaccine. Brit. Med. Jour., May 2, 1903, vol. i, pp. 1069-1074.

130. Wright. A note on the serum reaction of tubercle. Lancet, May 9, 1903, p. 1299.

131. Wright. A short treatise on anti-typhoid inoculation. An exposition of the principles of the method and a summary of the practical results achieved by the application of these principles. Practitioner, Jan.-March, 1904.

132. Wright. On the treatment of acne, furunculosis and sycosis by the therapeutic inoculations of staphylococcus vaccine. Brit. Med. Jour., May 7, 1904, p. 1075.

133. Wright. A lecture on the inoculation treatment of tuberculosis. Clinical Journal, 1904.

134. Wright. Pathological Suggestions. (1.) The preparation of microscopic slides for blood films. Lancet, July 9, 1904.

135. Wright. On the general principles of the therapeutic inoculation of bacterial vaccines as applied to the treatment of tubercular infection. Lancet, 1905, vol. ii, pp. 1598–1675.

136. Wright. Same article as 139. Med. Chir. Trans. Vol. lxxxix, 1905.

137. Wright. Zeit f. Hyg., Sept., 1903, vol. lxxii, p. 130.

138. Wright. The opsonic theory. Canadian Practitioner and Review, vol. xxi, 1906, p. 605.

139. Wright. The proceedings for testing and judging by the naked eye of the agglutination and bacteriolytic effects exerted by the sera of patients. Lancet, July 25, 1903, p. 214.

140. Wright. On certain new methods of blood examination, with some indications of their clinical importance. Lancet, Jan. 23, 1904, p. 215.

141. Wright and Douglas. On the action exerted upon the tubercle bacillus by human blood fluids, and on the elaboration of protective elements in the human organism in response to inoculation of a tubercle vaccine. Lancet, 1904, vol. ii, p. 1138.

142. Wright and Douglas. Same article as 145. Proc. Roy. Soc., vol. lxxiv, p. 159.

143. Wright and Douglas. An experimental investigation of the role of the blood fluids in connection with phagocytosis. Proc. Roy. Soc., vol. lxxii, 1903, pp. 357-370.

144. Wright and Douglas. Further observations on the role of the blood fluids in connection with phagocytosis. Proc. Roy. Soc., vol. lxxiii, 1904, pp. 128-141.

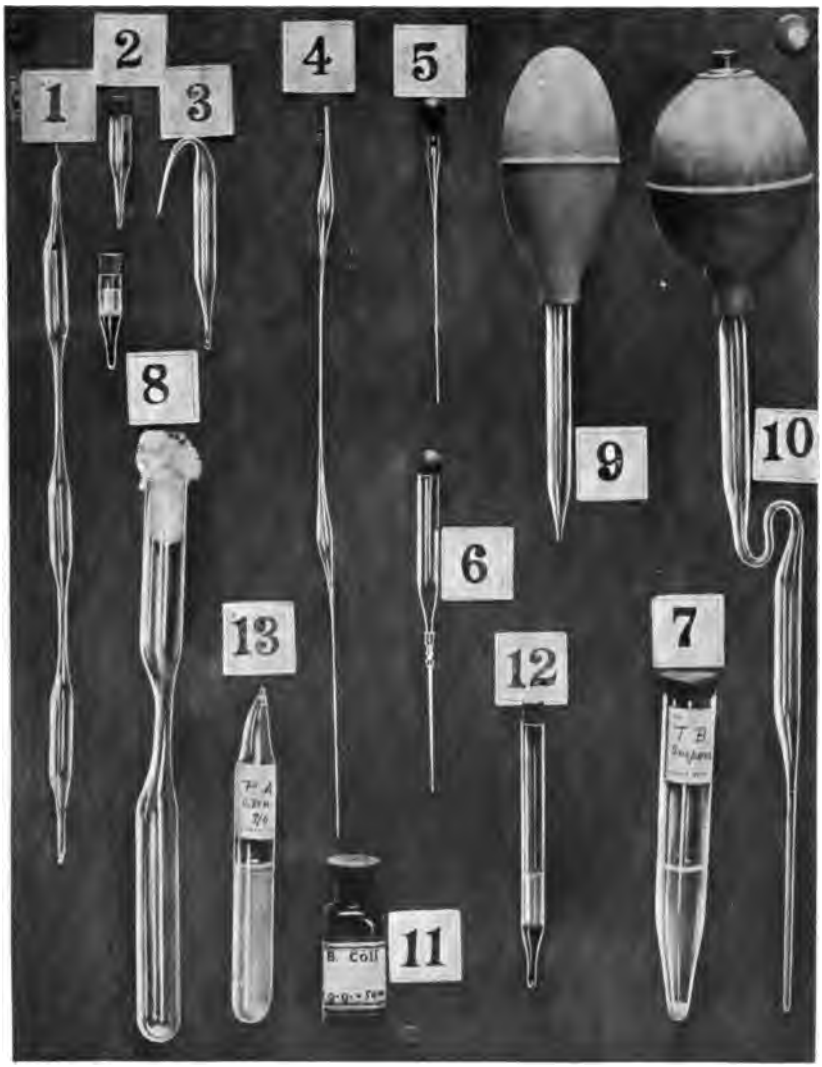
145. Wright and Douglas. On the action exerted upon the staphylococcus pyogenes by human blood fluids and on the elaboration of protective elements in the human organism in response to inoculations of a staphylococcus vaccine. Proc. Roy. Soc., vol. lxxiv, 1905, pp. 147-159.

146. Wright and Reid. On spontaneous phagocytosis, and on the phagocytosis which is obtained with the heated serum of patients who have responded to tubercular infection, or, as the case may be, to the inoculation of a tubercle vaccine. Proc. Roy. Soc., 1906, vol. lxxvii, pp. 211-225.

147. Wright and Reid. On the possibility of determining the presence or absence of tubercular infection by the examination of a patient's blood and tissue fluids. Proc. Roy. Soc., 1906, vol. lxxvii, pp. 194-211.

148. Wright and Reid. Notes on two cases of cholecystitis treated by inoculation of B. Coli. Br. Med. Jour., vol. i, 1906, p. 143.

149. Wright and Windsor. Jour. of Hyg., Oct., 1902, vol. ii, Nr. 4.



1. Tubing drawn into spindles for blood collecting tubes.
2. Blood collecting tubes, the lower containing blood.
3. Wright's blood collecting tube.
4. Tubing drawn out for mixing pipettes.
5. Mixing pipette.
6. Mixing pipette.
7. Centrifuge tube sealed with rubber cap for standard emulsions.
8. Heavy test tube drawn out for sealing (see No. 13).
9. Heavy tube drawn to fine capillary and supplied with bulb, for churning emulsion.
10. Wright's curly pipette.
11. Vaccine bottle with cap.
12. Tube for sedimenting corpuscles—corpuscular mass in bottom.
13. Bacterial suspension sealed in tube—for sterilization.

The Massachusetts Medical Society.

PROCEEDINGS.

CONTENTS OF APPENDIX.

	PAGE.
Proceedings of the Councillors, 1904–1905	1
Proceedings of the Society, Annual Meeting, June, 1905	10
Treasurer's Annual Report	19
Officers of the Society	22
Officers of the District Societies	28
Proceedings of the Councillors, 1905–1906	31
Proceedings of the Society, Annual Meeting, June, 1906	43
Treasurer's Annual Report	54
Officers of the Society	58
Officers of the District Societies	64
Proceedings of the Councillors, 1906–1907	67
Proceedings of the Society, Annual Meeting, June, 1907	80
Treasurer's Annual Report	89
Officers of the Society	93
Officers of the District Societies	99

The Massachusetts Medical Society.

PROCEEDINGS OF THE COUNCILLORS.

OCTOBER 5, 1904.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, October 5, 1904, at 12, noon.

The President, Dr. ARTHUR T. CABOT, in the chair.

The following Councillors were present:

Bristol North.
S. D. Presbrey.

Bristol South.
A. W. Buck,
H. G. Wilbur.

Essex North.
W. W. Pillsbury.

Essex South.
C. H. Bangs,
D. Coggin,
P. C. Proctor.

Franklin.
G. P. Twitchell.

Hampshire.
A. M. Belden.

Middlesex East.
J. P. Bixby,
E. S. Jack.

Middlesex North.
C. M. Frye,
T. F. Harrington,
W. P. Lawler,
J. V. Meigs.

Middlesex South.
D. E. Baker,
E. H. Bigelow.
R. E. Brown,
C. H. Cook,
F. G. Curtis,
J. G. Dearborn,
T. M. Durell,
W. Ela,
A. G. Griffin,
N. C. B. Haviland,
J. L. Hildreth,
E. D. Hooker,
H. B. McIntire,
H. E. Marion,
F. M. Sherman,
E. H. Stevens,
L. R. Stone,
R. Willis,
A. Worcester.

Norfolk.
F. R. Abbe,
F. P. Denny,
R. W. Hastings,
A. D. Hodges,
W. C. Kite,
J. H. S. Leard,
F. D. Lyon,
E. G. Morse,
W. Noyes,
W. T. Patch,
L. A. Roberts,
E. P. Starbird,
S. E. Stevens,
A. G. Williams,
A. M. Worthington,
E. C. Wylie.

Norfolk South.
C. S. Adams,
V. M. Tirrell.

Plymouth.
A. E. Paine.

Suffolk.
J. B. Ayer,
E. S. Boland,
E. G. Brackett,
B. M. Buckingham,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
A. Coolidge, Jr.,
F. W Draper,
R. H. Fitz,
C. F. Folsom,
C. M. Green,
B. J. Jeffries,
E. P. Joslin,
J. C. Munro,
A. H. Nichols,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
F. C. Shattuck,
G. B. Shattuck,
A. K. Stone,
O. F. Wadsworth,
J. C. Warren,
J. C. White,
G. Wolcott.

Worcester.
W. Davis,
G. E. Francis,
A. C. Getchell,
E. B. Harvey,
E. W. Norwood,
E. V. Scribner,
L. Wheeler,
A. Wood.

Worcester North.
J. R. Greenleaf,
S. E. Greenwood.

Total, 89.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

Vermont.—J. C. Munro, of Boston; F. P. Emerson, of Roxbury.

New York.—J. F. A. Adams, of Pittsfield; O. J. Brown, of North Adams.

New York State Medical Association.—C. F. Withington, of Boston; Homer Gage, of Worcester.

The Committee on Membership and Finance reported through Dr. Stone. In accordance with their recommendation, it was voted that the following be allowed to resign:

Edward R. Cutler, of Waltham.
Charles T. Fisher, of Princess Anne, Md.
Albert J. Roberts, of Bridgeport, Conn.

Also that the following be placed on the retired list:

George W. Gale, of East Saugus.

Also that the following be deprived of the privileges of membership under By-Law VIII. for non-payment of dues:

Edson M. Abbott, of Melrose.
Samuel D. Bartlett, of Lexington.
Russell H. Birge, of Cleveland, O.
George L. Black, of Lawrence.
Frederick G. Burnham, of Lyme, N. H.

Jeremiah A. Cronin, of South Boston.
Richard G. Eaton, of Holyoke.
George W. Ellison, of Central Falls, R. I.
Carl A. Ewald, of Deckerstown, N. J.
Edwin B. Flagg, of Worcester.
Edward W. Groll, of Boston.
Franklin W. Kenney, of Denver, Colo.
Henry LaMotte, of Salt Lake City, Utah.
Alexander L. McCormack, of East Boston.
John J. McGuigan, of Lynn.
William J. Moroney, of Boston.
Horace M. Nash, of Worcester.
Karl Ohnesorg, U. S. Navy.
George K. Roberts, of Attleborough.
Frank W. Sawyer, of Prescott, Ariz.
William Seaman, U. S. Navy.
John T. H. Slayter, U. S. Army.
Sumner P. Smith, of Lowell.
Henry G. Spooner, New York, N. Y.
Parker M. Ward, of Houlton, Me.
Walter L. Watson, of Biddeford, Me.
Jacob B. Wentworth, of Brownfield, Me.

Voted, That the following be restored to the privileges of membership:

George Wellesley Gates, of Roxbury.
Michael Ricard Donovan, of Lynn.

Adjourned at 12.25 P.M.

FRANCIS W. GOSS,
Recording Secretary.

FEBRUARY 1, 1905.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, February 1, 1905, at 12, noon.

The President, Dr. ARTHUR T. CABOT, in the chair.

The following Councillors were present:

Barnstable.	*Berkshire.*	*Bristol North.*
H. B. Hart.	L. C. Swift.	S. D. Presbrey.

Bristol South.
W. A. Dolan,
G. deN. Hough,
H. G. Wilbur.

Essex North.
J. A. Douglass,
J. G. McAllister,
W. W. Pillsbury.

Essex South.
C. H. Bangs.

Franklin.
G. P. Twitchell.

Hampden.
F. W. Chapin.

Hampshire.
A. M. Belden.

Middlesex East.
J. P. Bixby.

Middlesex North.
C. M. Frye,
G. H. A. Leahey,
J. V. Meigs,
J. B. O'Connor.

Middlesex South.
D. E. Baker,
R. E. Brown,
J. E. Cleaves,
C. H. Cook,
F. G. Curtis,
J. G. Dearborn,
W. Ela,
A. G. Griffin,
N. C. B. Haviland,
E. G. Hoitt,
E. D. Hooker,
H. E. Marion,
J. T. G. Nichols,
C. E. Prior,
F. M. Sherman,
E. H. Stevens,
L. R. Stone,
H. P. Walcott,
R. Willis.

Norfolk.
E. E. Bancroft,
B. H. Calkins,
G. H. French,
D. Graham,
A. D. Hodges,
F. D. Lyon,
W. T. Patch,
L. A. Roberts,
E. P. Starbird,
S. E. Stevens,
A. G. Williams,
A. M. Worthington,
E. C. Wylie.

Norfolk South.
V. M. Tirrell.

Plymouth.
N. K. Noyes,
A. E. Paine.

Suffolk.
J. B. Ayer,
E. S. Boland,
H. L. Burrell,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
R. H. Fitz,
C. F. Folsom,
C. M. Green,
F. B. Harrington,
E. P. Joslin,
J. H. McCollom,
A. B. Morong,
J. G. Mumford,
J. C. Munro,
A. H. Nichols,
C. B. Porter,
A. Post.
J. M. Putnam,
W. L. Richardson,
G. B. Shattuck,
A. K. Stone,
H. F. Vickery,
O. F. Wadsworth,
J. C. Warren,
J. C. White,
H. Williams,
G. Wolcott.

Worcester.
W. Davis,
O. H. Everett,
G. E. Francis,
E. B. Harvey,
H. M. Quinby.

Worcester North.
C. H. Rice.

Total, 87.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other Medical Societies:

American Medical Association, 1905 and 1906.—J. F. A. Adams, of Pittsfield; H. L. Burrell, R. H. Fitz, of Boston.

Alternates.—J. L. Hildreth, of Cambridge; C. H. Williams, of Boston; Homer Gage, of Worcester.

Maine.—Alfred Worcester, of Waltham; W. P. Bowers, of Clinton.

New Hampshire.—D. N. Blakely, of Roxbury; Leonard Huntress, of Lowell.

Rhode Island.—L. M. Palmer, of Framingham; W. N. Swift, of New Bedford.

Connecticut.—Leonard Wheeler, of Worcester; G. A. Bancroft, of Natick.

New Jersey.—E. P. Joslin, of Boston; H. E. Marion, of Brighton.

The following Committee was appointed:

To Audit the Treasurer's Accounts.—Alexander Quackenboss, M. P. Smithwick.

The Committee on Membership and Finance reported through Dr. Stone. In accordance with their recommendation it was voted that the following be allowed to resign:

Noah A. Burr, of South Manchester, Conn.
Charles F. Morse, of U. S. Army.
Walter N. Sharp, of Indianapolis, Ind.

Also, that the following be placed on the retired list:

Winthrop Butler, of Vineyard Haven.
George W. Doane, of Hyannis.
Samuel Pitcher, of Hyannis.

The President nominated and it was voted that Dr. D. D. Gilbert of Dorchester be appointed to fill the vacancy in the Committee on State and National Legislation caused by the death of Dr. S. W. Abbott.

Adjourned at 12.45 P.M.

FRANCIS W. GOSS,
Recording Secretary.

June 13, 1905.

THE ANNUAL MEETING of the Councillors was held in the Medical Library, Boston, on Tuesday, June 13, 1905, at 5 P.M.

The President, Dr. ARTHUR T. CABOT, in the chair.

The following Councillors were present:

Berkshire.
O. J. Brown.

Bristol North.
A. S. Deane,
W. Y. Fox,
S. D. Presbrey.

Bristol South.
A. W. Buck,
W. A. Dolan,
G. deN. Hough,
H. G. Wilbur.

Essex North.
I. J. Clarke,
C. E. Durant,
J. G. McAllister,
E. H. Noyes.

Franklin.
H. G. Stetson,
G. P. Twitchell,
N. P. Wood.

Hampden.
T. S. Bacon,
H. C. Emerson,
G. E. Fuller,
D. E. Keefe.

Hampshire.
J. M. Fay.

Middlesex East.
C. Dutton,
G. N. P. Mead.

Middlesex North.
C. M. Frye,
T. F. Harrington,
W. A. Johnson,
W. P. Lawler,
G. H. A. Leahey,
J. V. Meigs.

Middlesex South.
E. H. Bigelow,
T. Chamberlin,
J. M. Crocker,
T. M. Durell,
W. Ela,
G. W. Gay,
N. C. B. Haviland,
J. L. Hildreth,
J. A. McDonald,
H. B. McIntire,
H. E. Marion,
G. A. Miles,
J. T. G. Nichols,
G. Ryder,
F. M. Sherman,
F. G. Smith,
J. E. Somers,
E. H. Stevens,
L. R. Stone,
F. R. Stubbs,
W. H. Sylvester,
F. W. Taylor,
J. Tolman,
H. P. Walcott,
R. Willis,
H. A. Wood,
A. Worcester.

Norfolk.
B. H. Calkins,
S. Crowell,
F. H. Davenport,
F. W. Draper,
D. D. Gilbert,
D. Graham,
R. W. Hastings,
J. H. S. Leard,
F. D. Lyon,
E. G. Morse,
W. Noyes,
A. P. Perry,
L. A. Roberts,
E. P. Starbird,
H. R. Stedman,
S. E. Stevens,
W. N. Tenney,
E. Thayer,
W. A. White,
A. M. Worthington,
E. C. Wylie.

Norfolk South.
O. H. Howe,
N. S. Hunting,
F. E. Jones,
E. N. Mayberry.

Plymouth.
H. W. Dudley,
A. E. Paine,
F. G. Wheatley.

Suffolk.
J. B. Ayer,
E. G. Brackett,
E. H. Bradford,
E. M. Buckingham,
H. L. Burrell,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
E. G. Cutler,
H. Derby,
R. H. Fitz,
C. F. Folsom,
M. F. Gavin,
C. M. Green,
F. B. Harrington,
B. J. Jeffries,
F. I. Knight,
J. H. McCollom,
A. B. Morong,
J. G. Mumford,
A. H. Nichols,
C. B. Porter,
A. Post,
J. J. Putnam,
J. M. Putnam,
W. L. Richardson,

F. C. Shattuck,
G. B. Shattuck,
A. K. Stone,
H. F. Vickery,
G. Wolcott.

Worcester.
P. P. Comey,
W. Davis,
J. T. Duggan,
G. E. Francis,
A. C. Getchell,
E. B. Harvey,
E. W. Norwood,
L. Wheeler,
S. B. Woodward.

Worcester North.
C. E. Bigelow,
E. P. Miller,
C. H. Rice,
E. A. Sawyer,
F. H. Thompson.

Total, 128.

The record of the last meeting was read and accepted.

The Secretary read the names of new and of deceased Fellows.

The Treasurer, Dr. Buckingham, read his annual report.

The Auditing Committee reported that they found the accounts correctly cast and properly vouched, and that the Treasurer has in his possession the securities called for.

Voted, To accept the Treasurer's report.

The Committee on Membership and Finance reported through Dr. Stone, and recommended that $4,500.00 of the surplus in the treasury be distributed among the District Societies.

Voted, That the recommendation be adopted.

In accordance with the recommendation of the same Committee it was voted, that the resignations of the following be accepted:

Harrie S. Baketel, of Jersey City, N. J.
Allston F. Hunt, of Swampscott.
Charles H. Kingsbury, of Danielson, Conn.
Mary E. Newcomb, of Blissfield, Mich.
Richard R. Pettigrew, of Scranton, Pa.
Thomas G. Reed, of South Boston.

Also, that the following be placed on the retired list:

Willard S. Everett, of Waban.
James Hedenberg, of Medford.
William H. Lathrop, of Lowell.
Charles P. Morrill, of North Andover.
William Neilson, of Marblehead.

The Committee on Publications presented their report.

The Committee on Medical Diplomas reported through Dr. Marion, and presented a revised list of Medical Colleges whose

diplomas they recommended should be recognized for admission to the Society.

Voted, To adopt the list presented by the Committee.

The Committee on State and National Legislation reported:

That they have assisted in opposing the Osteopathic bill, the Optometry bill, and the Anti-vivisection bills, all of which were killed in the Committees of the Legislature to which they were referred.

They obtained the passage of an act regulating the sale and possession of wood alcohol.

They recommend the appointment of an auxiliary Legislative Committee, to consist of one member from each District of the State, similar to the auxiliary Legislative Committee of the American Medical Association.

Voted, To accept the report, and that the Committee be empowered to appoint the auxiliary Committee referred to therein.

The Librarian, Dr. Brigham, presented his annual report.

The Committee on Nominations reported a list of candidates for the offices of the Society for the ensuing year, and the same were elected by ballot:

President . .	ARTHUR T. CABOT, of Boston.
Vice-President .	FREDERICK H. THOMPSON, of Fitchburg.
Treasurer .	EDWARD M. BUCKINGHAM, of Boston.
Corresponding Sec'y	CHARLES W. SWAN, of Brookline.
Recording Secretary	FRANCIS W. GOSS, of Roxbury.
Librarian .	EDWIN H. BRIGHAM, of Brookline.

JOHN L. HILDRETH, of Cambridge, was chosen Orator for the annual meeting of the Society in 1906.

Voted, That the next annual meeting of the Society be held in Boston on the second Wednesday in June, 1906.

The following Standing Committees were appointed:

Of Arrangements.

Farrar Cobb, Lincoln Davis,
A. P. Perry, L. W. Gilbert,
G. S. C. Badger, C. C. Simmons.

On Publications.

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

On Membership and Finance.

L. R. Stone, F. W. Goss, Walter Ela,
C. M. Green, A. Coolidge, Jr.

To Procure Scientific Papers.

C. F. Withington, J. B. Blake, B. P. Croft,
R. C. Larrabee, M. D. Clarke.

On Ethics and Discipline.

C. G. Carleton, Edward Cowles, J. F. A. Adams,
Leonard Wheeler, J. A. Gage.

On Medical Diplomas.

H. E. Marion, O. F. Rogers, H. W. Newhall.

On State and National Legislation.

A. T. Cabot, H. P. Bowditch, S. D. Presbrey,
G. G. Sears, D. D. Gilbert.

Voted, That Dr. William W. Eaton, of Danvers, be restored to active membership.

Adjourned at 6.10 P.M.

FRANCIS W. GOSS,
Recording Secretary.

The Massachusetts Medical Society.

PROCEEDINGS OF THE SOCIETY.

ANNUAL MEETING.

First Day.

June 13, 1905.

Sectional meetings were held in the Medical Library, Boston, on Tuesday, June 13, 1905, at 2 P.M.

The sections were organized and papers were presented as follows:

SECTION IN MEDICINE.

Dr. George W. Gay, of Newton . . . . . Chairman.
Dr. Ralph C. Larrabee, of Boston . . . . . Secretary.

Some Interesting Features connected with the Outbreak of Small Pox at North Adams, Mass., in 1904.—By Dr. L. A. Jones, of North Adams.

Ocular Vertigo, of Interest to the General Practitioner.—By Dr. B. P. Croft, of Greenfield.

A Clinical and Laboratory Study of the Therapeutic Value of Hydrochloric Acid in Diseases of the Stomach.—By Dr. R. F. Chase, of Brookline.

The Medical Treatment of Gastric Ulcer and Hyperchlohydria.—By Dr. F. C. Shattuck, of Boston.

Methods in Medicine.—By Dr. Lewellys F. Barker, of Chicago, Ill.

This address was introductory to the demonstration given on the following day.

SECTION IN SURGERY.

Dr. GEORGE H. MONKS, of Boston . . . . . Chairman.
Dr. J. BAPST BLAKE, of Boston . . . . . Secretary.

THE APPLIED ANATOMY OF THE FRONTAL SINUSES.—By Dr. H. P. Mosher, of Boston.

EMPYEMA OF THE FRONTAL SINUS.—By Dr. F. C. Cobb, of Boston.

WHAT THE SUBURBAN SURGEON IS DOING IN THE ABDOMEN, AND HOW HE DOES IT.—By Dr. C. E. Durant, of Haverhill.

EARLY DIAGNOSIS OF SURGICAL DISEASE IN THE URINARY ORGANS.—By Dr. Benjamin Tenney, of Boston.

CAUSES OF DISABILITY AFTER FRACTURES OF THE LOWER LEG AND ANKLE.—By Dr. F. J. Cotton, of Boston.

At 8 P.M., the Shattuck Lecture was delivered by RUSSELL H. CHITTENDEN, Ph.D., of New Haven, Conn.

Following the lecture a reception was given to the President by the Society.

SECOND DAY.

JUNE 14, 1905.

THE Society met in Mechanic Building, Boston, on Wednesday, June 14, 1905, at 9 A.M., for the exercises of the one hundred and twenty-fourth Anniversary.

The President, Dr. ARTHUR T. CABOT, in the chair.

The record of the last annual meeting was read and accepted.

In accordance with notice given at the last annual meeting, on motion of Dr. FitzHugh, it was

Voted, That so much of Rule I. of the Rules and Orders of the Society as calls for the reporting of names of new Fellows be annulled.

The Secretary announced that 171 new Fellows had been admitted during the past year and that 37 deaths had been recorded.

The following are the lists:—

Admissions reported since June 7, 1904.

1905	Abbott, Harry Daniel	Danvers.
1904	Andrews, Robert Eaton	Cambridge.
1905	Ash, Thomas Francis	Rockland.
1904	Atwood, Abel Wilson	Mount Hermon.
1904	Bail, John Warren	Brookline.
1905	Bailey, Frederick James	Dorchester.
1905	Barney, James Dellinger	Boston.
1904	Barrett, Michael Francis	Brockton.
1905	Bastian, George Leon	Lowell.
1904	Bellamy, William Woolsey	Dorchester.
1904	Binford, Ferdinand Augustus	Hyannis.
1904	Blake, Allen Hanson	Cristobal, C. Z.
1904	Blenkhorn, James	Stoneham.
1905	Bliss, George Stephen	Waverley.
1905	Bonoff, Zelly Adam	Worcester.
1905	Boutwell, Horace Keith	Boston.
1904	Brown, George Christopher	Worcester.
1904	Bürger, Gothfried Leonhard Hagen	Boston.
1905	Burke, Francis Ramon	Quincy.
1905	Burnham, Parker	Gloucester.
1905	Butler, Patrick Francis	South Boston.
1904	Butterfield, George Kittredge	Taunton.
1904	Byrnes, Harry Francis	Springfield.
1904	Cannon, Walter Bradford	Cambridge.
1905	Chandler, Clarence Luther	Fitchburg.
1905	Chapman, Howard Jones	Everett.
1905	Chesley, Alfred Ervan	Lawrence.
1905	Clark, George Oliver	Boston.
1904	Clarke, Inez Louise	Cambridge.
1904	Closson, Gershom Loveland	Wakefield.
1905	Cook, Philip Howard	Worcester.
1905	Cooley, Abbott Lathrop	Chicopee Falls.
1904	Coolidge, Frederick Shurtleff	Pittsfield.
1905	Coulson, Richard	Somerville.
1904	Cort, Parker Martin	Springfield.
1904	Cummings, John Joseph	Worcester.
1904	Dailey, Edward Joseph	Somerville.
1904	Dalton, John Edward	Warren.
1905	Daly, Jeremiah James	North Andover.
1904	Darling, Byron Clary	Boston.
1904	Devere, Fred Hewitt	Sterling.
1905	Dexter, Fred Fay	Granby.
1904	Donoghue, John Joseph	Clinton.

1904	Drury, Dana Warren	Lowell.
1905	Easton, Charles Daniel	Boston.
1904	England, Albert Charles	Pittsfield.
1905	Everett, Frederick Luther	Springfield.
1904	Fairing, John Walker	Palmer.
1904	Ferrin, William Warren	Brookline.
1904	Finkelstein, Harry	Boston.
1904	Fischbein, Louis	Boston.
1904	Fish, Louis	Fitchburg.
1905	Fitch, Ralph Roswell	Dorchester.
1904	Fitzgibbon, Edward James	Dorchester.
1904	Fleming, Patrick Joseph	Cambridge.
1905	Flett, Penelope McNaughton	Waverley.
1905	Floyd, Cleaveland	Boston.
1904	Foster, Ellis Edwin	New Bedford.
1904	Fountain, Oliver Reynolds	Roxbury.
1905	Freedman, Louis Mark	Chelsea.
1905	Gafney, Harry Dabol	Ware.
1905	Garland, Roy	Boston.
1905	Gay, Clarence Bertram	Fitchburg.
1904	Greene, Ransom Alphonzo	Monson.
1905	Greenwood, Arthur Moses	Boston.
1904	Grimes, Loring	Lynn.
1904	Grover, Arthur Leon	Hyde Park.
1905	Hamilton, Robert De Lancey	Newburyport.
1905	Harper, Alfred Alphonso	North Adams.
1905	Hartwell, John Bryant	Boston.
1904	Haviland, Walter Childs	Worcester.
1905	Hayes, Justin Edward	Chesterfield.
1905	Hedin, Carl Johan	Tewksbury.
1904	Hill, George Jackson	Beverly.
1904	Hill, Thomas Chittenden	Boston.
1904	Holden, Nebuther	Southampton.
1904	Homans, John	Boston.
1904	Howe, Harry Newell	Greenfield.
1905	Hussey, Edward John	Boston.
1905	Jackson, John Patrick	Fall River.
1904	Keith, Halbert Lynn	Milford.
1905	Kelly, William Dugan	Boston.
1905	Kenney, Walter Clement	Tewksbury.
1905	Kent, Ralph Porter	Attleboro'.
1904	Kerr, Isabella Dickieson	Boston.
1905	Kimpton, Arthur Ronald	Somerville.
1904	Kirby, Holder Crary	New Bedford.
1904	Kirby, James Richard	Clinton.
1904	Lang, Herbert Boardman	Dorchester.
1905	Larrabee, Herbert Manson	Tewksbury.

1905	Lee, William George	Boston.
1904	Levins, Nathan Noah	Boston.
1905	Linenthal, Harry	Roxbury.
1904	Little, John Mason	Boston.
1905	MacAusland, William Russell	Boston.
1905	McCaffrey, Charles Francis	Somerville.
1905	MacCormick, John Allan	Boston.
1904	MacCoy, William Edward	Brookline.
1904	McCullough, Edward Aloysius	Worcester.
1904	McGillicuddy, Richard Aloysius	Turner's Falls.
1905	Mackillop, Daniel	Cambridge.
1904	McMann, William Henry	Jamaica Plain.
1905	McPherson, George Edwin	South Boston.
1905	MacQuaid, John Stephen	Springfield.
1904	Madden, William Daniel	Boston.
1904	Mahoney, Daniel Francis	Boston.
1905	Makechnie, Arthur North	Cambridge.
1904	Marshall, Herman Prince	Quincy.
1905	Mayberry, Frank Eugene	Boston.
1905	Meehan, Patrick Joseph	Lowell.
1905	Mellus, Edward	Worcester.
1905	Mendelsohn, Louis	Dorchester.
1905	Messinger, Harry Carleton	South Boston.
1904	Moore, George Colton	Boston.
1904	Moriarity, James	Athol.
1905	Murphy, Thomas William	Lawrence.
1905	Myers, Edward Everett	Roxbury.
1904	Myles, Leo Thomas	Worcester.
1904	Ober, Ralph Beverley	Springfield.
1905	O'Brien, Loretta Joy	Boston.
1904	Ordway, Clarence Eugene	Winchester.
1905	Osgood, George	Boston.
1904	Palfrey, Francis Winslow	Boston.
1905	Parr, John	Methuen.
1905	Patterson, Alice Maria	Peabody.
1904	Paull, Chester Alpheus	Sherborn.
1904	Peters, William Chute	Bangor, Me.
1904	Phippen, Walter Gray	Salem.
1905	Pike, Forrest Fay	Melrose.
1904	Pope, Ernest Folger	Worcester.
1905	Powers, George Herman, Jr.	Boston.
1905	Quessy, Alfred Henry	Fitchburg.
1905	Reardon, Daniel Bartholomew	Quincy.
1904	Richardson, Frank Linden	Boston.
1904	Ripley, Horace Greeley	Taunton.
1904	Ritter, Henry	Springfield.
1904	Rochette, Edward Charles	Worcester.

1904	Rowland, Russell Sturgis	Detroit, Mich.
1905	Rumrill, Samuel Dudley	Springfield.
1904	Safford, Wilber Pray	Brockton.
1904	Sanger, Guy Edward	Arlington.
1905	Sargent, Walter Leslie	Quincy.
1905	Scanlan, Thomas John	Boston.
1905	Segur, Willard Blossom	Enfield.
1905	Seymour, Frederick Ward	Boston.
1904	Seymour, Malcolm	Boston.
1904	Shaw, John Port	Brockton.
1905	Sheehan, William Joseph	South Boston.
1905	Simmons, Fred Albert	Brockton.
1905	Sinclitico, Guiseppe	Lawrence.
1904	Slack, Francis Hervey	Boston.
1904	Spaid, Charles Jacob	Springfield.
1904	Starbuck, Joseph Clinton	North Easton.
1905	Stoneman, Edgar Amos	Springfield.
1904	Stoodley, Harry Marr	Somerville.
1905	Topaz, Anna	Boston.
1904	Torbert, James Rockwell	Boston.
1904	Treichler, Albert Julius	Springfield.
1904	Vincent, Beth	Boston.
1905	Vrooman, Earle Morey	Cummington.
1904	Wald, Rudolf Henry	Salem.
1905	Wallace, Anna Marie	Waverley.
1904	Ward, Roy Joslyn	Worcester.
1904	Wernick, Benzion G.	Boston.
1905	Whitney, Edward William	Ware.
1905	Whitney, Harvey Herbert	Holyoke.
1904	Whiton, Ross Kittredge	Quincy.
1904	Winchester, George Wesley	Mattapan.
1905	Woodbury, Willard Porter	Beverly.
1904	Youngman, Julia Ross	Boston.
1904	Zabriskie, Fred Templeton	Pittsfield.

Total, 171.

List of Deceased Fellows.

Admitted.	Name.	Residence.	Date of Death.	Age.
1841	Abbot, Samuel Leonard	Boston	July 1, 1904	86
1862	Abbott, Samuel Warren	Newton	Oct. 22, 1904	67
1880	Allen, Franklin Haley	Haverhill	April 23, 1905	49
1849	Babbitt, Henry Smith	Dorchester	Dec. 10, 1904	78
1857	Bemis, Merrick	Worcester	Oct. 3, 1904	84
1884	Birtwell, Charles Ebenezer	Lawrence	Dec. 8, 1904	43
1866	Breck, Theodore Frelinghuysen	Springfield	June 26, 1904	59
1899	Brown, Anthony Leopold	Springfield	April 22, 1905	42

1902	Brown, Henry Rienzi	Leominster	Feb.	16,	1905	63
1883	Canfield, Ralph Metcalfe	Boston	Nov.	3,	1904	45
1900	Creesy, Everett Larcom	Beverly	May	28,	1905	30
1897	Donlan, John Malachi	Fitchburg	Jan'y	26,	1905	30
1889	Draper, Joseph Rutter	Westford	Oct.	30,	1904	42
1894	Drown, Edward Payson	Malden	May	25,	1905	39
1863	Flowers, William Caldwell	Cambridge	Oct.	20,	1904	72
1901	Goodrich, Harriet Rawson	Salem	Aug.	23,	1904	34
1858	Gould, Joshua Bayley	Somerville	Dec.	19,	1904	85
1867	Greenough, Francis Boott	Boston	Oct.	16,	1904	65
1873	Healey, James Joseph	Newburyport	Nov.	11,	1904	55
1870	Hunt, Israel Thorndike	Charlestown	Feb.	16,	1905	63
1848	Hunt, Otis Eugene	Newton	Jan'y	20,	1905	82
1866	Johnson, Otis Henry	Haverhill	July	16,	1904	63
1890	King, William Rufus	Boston	Feb.	17,	1905	37
1888	Lombard, John Patrick	Dorchester	March	21,	1905	44
1900	McNiff, William Joseph	Pepperell	June	17,	1904	
1887	Mahoney, John Bernard	Medford	April	20,	1904	41
1854	Newhall, Edward	Lynn	June	12,	1905	83
1874	Newton, Adin Hubbard	Everett	July	7,	1904	87
1890	Padula, Thomas Francis	Quincy	Feb.	9,	1905	45
1875	Phelps, Rollin Horace	Littleton	Jan'y	28,	1905	71
1896	Steele, John McClary	Worcester	Aug.	1,	1904	37
1890	Swift, Lawrence Chew	Pittsfield	June	1,	1905	53
1871	Thayer, Samuel Ezra	Wenham	Feb.	5,	1905	62
1866	Vaughan, Charles Everett	S't.Barbara, Cal.	June	24,	1904	68
1897	Vincent, Philip	Waltham	April	30,	1903	45
1887	Warren, William Barnard	Groton	April	29,	1905	51
1849	Webber, Alonzo Carter	Cambridge	Aug.	5,	1904	77

Total, 37

The Treasurer's report was read.

The records of the Boards of Trial in the following cases were read.

Voted, That the sentences of the Boards of Trial be imposed, and that

Howard K. Glidden, of Lynn,
Seraph Frissell, of Springfield,
Charles A. Eastman, of Old Orchard, Me.,

be and are expelled from their membership of The Massachusetts Medical Society.

Following the business meeting there was given until 12 o'clock in a number of booths

A DEMONSTRATION OF CLINICAL AND LABORATORY METHODS USED IN MEDICINE.

The demonstration was organized by Professor W. T. Councilman, and was conducted as follows:

Drs. C. J. Blake.—Methods of Diagnosis in Diseases of the Ear.

J. B. Blake and C. A. Porter.—Methods of Physical Diagnosis in Surgery.

J. T. Bowen.—Methods of Diagnosis in Skin Diseases.

R. C. Cabot.—Methods of Physical Diagnosis in Internal Medicine.

H. A. Christian.—Demonstration in Pathological Anatomy.

E. A. Codman.—Methods in Diagnosis by means of X-ray.

Algernon Coolidge, Jr.—Methods of Diagnosis in Diseases of the Nose and Throat.

R. L. Emerson.—Methods in Examination of Urine.

H. C. Ernst.—Methods in Bacteriology.

A. H. Gould.—Methods in Estimating Coagulation Time of Blood.

R. C. Larrabee and A. E. Austin.—Methods in Examination of Blood.

Timothy Leary.—Methods in Legal Medicine.

F. T. Lord.—Methods in Examination of Sputum.

F. T. Murphy.—Methods of Study of Blood Pressure.

Percy Musgrave.—Methods in Cyto-Diagnosis.

Franz Pfaff and E. P. Joslin.—Methods in Gastric Diagnosis.

J. H. Pratt.—Methods in Examination of Feces.

J. J. Putnam.—Methods in Neurology.

Theobald Smith and H. W. Hill.—Methods used in Board of Health Examinations.

Paul Thorndike.—Methods of Diagnosis in Genito-Urinary Diseases.

J. H. Wright.—Methods of Rapid Anatomical Diagnosis.

At 12 M. the Annual Discourse was delivered by Dr. CHARLES A. DREW, of State Farm, Bridgewater.

Voted, That the thanks of the Society be presented to the Orator for his able and interesting address.

Voted, That the Massachusetts General and Boston City Hospitals be thanked for their interest and efficient aid in the Demonstrations of Methods used in Medicine.

Voted, That Dr. Baker be thanked for his very instructive address given yesterday.

The following delegates from other State Medical Societies were present:

Maine.—Dr. Hiram Hunt.
New Hampshire.—Dr. G. P. Conn.
Vermont.—Dr. F. R. Stoddard.
Rhode Island.—Dr. W. C. Munroe.
Connecticut.—Dr. O. C. Smith.

At 1 P.M. the Annual Dinner was served to more than eleven hundred Fellows.

FRANCIS W. GOSS,
Recording Secretary.

TREASURER'S REPORT.

The accounts for the year ending April 15, 1905, are as follows:

Receipts.

Balance from the previous year		$9,984 87	
Assessments to District Treasurers —			
Barnstable	$130 00		
Berkshire	325 00		
Bristol North	225 00		
Bristol South	485 00		
Essex North	440 00		
Essex South	715 00		
Franklin	180 00		
Hampden	635 00		
Hampshire	285 00		
Middlesex East	260 00		
Middlesex North	495 00		
Middlesex South	1,520 00		
Norfolk	1,380 00		
Norfolk South	205 00		
Plymouth	315 00		
Suffolk	2,600 00		
Worcester	1,040 00		
Worcester North	320 00		
	$11,555 00		
Assessments at Annual Meeting	925 00		
Assessments to Treasurer	490 00		
Total Assessments	$12,970 00	12,970 00	
Sale of Dinner Tickets		12 00	
Interest Bay State Trust Co.	$123 61		
Interest New England Trust Co.	103 17		
Total Int. on funds subject to check	$226 78	226 78	
Int. Franklin Savings Bank	37 93		
Int. Suffolk Savings Bank	35 30		
Int. Roxbury Inst. for Savings	35 30		
Int. Provident Inst. for Savings	35 30		
Total Interest Savings Banks	$143 83	143 83	
Interest United States Bond	400 00		
Interest Massachusetts Bonds	175 00		
Total Interest on Bonds	$575 00	575 00	
Interest Annuity Mass. Hospital Life Ins. Co.		816 80	
Premiums on check		10	
Total receipts			$24,729 38

Expenses.

Secretary's Expense: —		
Printing and postage	$168 07	
Clerk	16 80	
Engrossing	13 90	
Stenographer	18 00	
Stationery	2 25	
Incidentals	3 08	
	$222 10	222 10
Librarian's Expense: —		
Delivering Communications in Metropolitan Districts	$73 10	
Printing, wrappers and postage	111 60	
Stationery	8 00	
	$192 70	192 70
Censors' Expense		466 07
Supervisors' Expense		14 45
Treasurer's Expense: —		
Card catalogue for ledger	$9 65	
Stationery, printing and postage	88 40	
Clerk hire at Annual Meeting	25 00	
Transporting accounts to and from meeting	3 50	
Box Bay State Trust Co.	10 00	
	$136 55	136 55
District Treasurers' Expense and Commissions		705 35
Exchange		1 00
Overpaid assessments		15 00
Salaries		1,400 00
Rent		750 00
Annual Meeting: —		
Rent of hall	$511 50	
Stationery, printing and postage	77 65	
Clerical work for committee	17 50	
Stereopticon	21 00	
Stenographer	5 00	
Card signs	7 40	
Towel Supply Co.	7 01	
Service of coat room	5 50	
Police	12 00	
Florist	50 00	
Music	40 00	
Caterer	2,497 46	
Cigars	161 50	
Incidentals	19 26	
	$3,432 78	3,432 78

Shattuck Lecture: —			
Lecturer	$200 00		
Advertising	1 38		
	$201 38	201 38	
Cotting Lunch		99 00	
Annual Dividend		4,500 00	
Committee To Procure Scientific Papers		13 10	
Committee On Publications		1,284 02	
Committee On Ethics		23 00	
Committee On Diplomas		12 18	
Boards of Trial		56 65	
Annual Meeting of 1905, tickets		5 00	
Total Expenses		13,530 33	13,530 33
Balance			$11,199 05
This balance is represented by			
Deposit in Bay State Trust Co.		$6,335 21	
Deposit in New England Trust Co.		5,361 04	
Undrawn interest in Suffolk Savings Bank		35 30	
Undeposited check		5 00	
		$11,736 55	11,736 55
From which deduct checks still out			537 50
Balance			$11,199 05

There has been no change in the permanent investments of the Society. They are as follows: —

Deposit Roxbury Institution for Savings (Cotting Fund)	$1,000 00
Deposit Provident Institution for Savings (Cotting Fund)	1,000 00
Deposit Suffolk Savings Bank (Cotting Fund)	1,000 00
United States 4 per cent. Bond (Phillips Fund)	10,000 00
Annuity Mass. Hospital Life Ins. Co. (Shattuck Fund)	9,166 87
Annuity Mass. Hospital Life Ins. Co. (Permanent Fund)	11,253 30
Massachusetts 3½ per cent. gold bonds (Permanent Fund)	5,000 00
Deposit Franklin Savings Bank (Permanent Fund)	1,074 48
Total,	$39,494 65

EDWARD M. BUCKINGHAM,
Treasurer.

BOSTON, MAY 9, 1905.

The undersigned, a duly appointed committee, having examined the books of the treasurer, find them correctly cast and properly vouched, and also that he has in his possession the securities called for.

ALEX. QUACKENBOSS.
M. P. SMITHWICK.

Officers of the Massachusetts Medical Society.

1905—1906.

CHOSEN JUNE 13, 1905.

ARTHUR T. CABOT,	Boston,	PRESIDENT.
FREDERICK H. THOMPSON,	Fitchburg,	VICE-PRESIDENT.
EDWARD M. BUCKINGHAM,	Boston,	TREASURER.
CHARLES W. SWAN,	Brookline,	COR. SECRETARY.
FRANCIS W. GOSS,	Roxbury,	REC. SECRETARY.
EDWIN H. BRIGHAM,	Brookline,	LIBRARIAN.

Standing Committees.

Of Arrangements.

FARRAR COBB, LINCOLN DAVIS,
A. P. PERRY, L. W. GILBERT,
G. S. C. BADGER, C. C. SIMMONS.

On Publications.

O. F. WADSWORTH, G. B. SHATTUCK, H. L. BURRELL.

On Membership and Finance.

L. R. STONE, F. W. GOSS, WALTER ELA.
C. M. GREEN, A. COOLIDGE, JR.

To Procure Scientific Papers.

C. F. WITHINGTON, J. B. BLAKE, B. P. CROFT,
R. C. LARRABEE, M. D. CLARKE.

On Ethics and Discipline.

C. G. CARLETON, EDWARD COWLES, J. F. A. ADAMS,
LEONARD WHEELER, J. A. GAGE.

On Medical Diplomas.

H. E. MARION, O. F. ROGERS, H. W. NEWHALL.

On State and National Legislation.

A. T. CABOT, H. P. BOWDITCH, S. D. PRESBREY,
G. G. SEARS, D. D. GILBERT.

Presidents of District Societies—Vice-Presidents (Ex-Officiis).

[Arranged according to seniority.]

Abner Post,	G. B. Underwood,
H. G. Leslie,	E. W. Norwood,
G. W. Clement,	S. H. Parks,
W. M. E. Mellen,	T. J. Robinson,
J. F. Welch,	I. O. Cummings,
Amasa Howard,	J. J. Flynn,
W. P. Chisholm,	C. A. Pratt,
C. C. Messer,	C. F. Branch,
Alfred Worcester,	C. H. Bangs.

Councillors.

Barnstable.—W. D. Kinney, Osterville; E. M. Parker, South Yarmouth.

Berkshire.—J. F. A. Adams, Pittsfield; O. J. Brown, H. Bushnell, North Adams; W. W. Leavitt, Pittsfield; A. T. Wakefield, Sheffield.

Bristol North.—J. W. Battershall, Attleboro'; A. S. Deane, W. Y. Fox, S. D. Presbrey, Taunton.

Bristol South.—A. W. Buck, W. A. Dolan, S. V. Merritt, H. G. Wilbur, Fall River; C. F. Connor, G. deN. Hough, F. E. Stetson, A. P. Webber, New Bedford.

Essex North.—G. M. Atwood, Bradford; J. A. Douglass, Amesbury; I. J. Clarke, C. E. Durant, Haverhill; J. G. McAllister, Lawrence; E. H. Noyes, W. W. Pillsbury, Newburyport; R. B. Root, Georgetown; G. B. Sargent, Lawrence.

Essex South.—F. S. Atwood, Salem; F. W. Baldwin, Danvers; C. M. Cobb, Lynn; D. Coggin, Salem; S. P. F. Cooke, Gloucester; H. K. Foster, Peabody; C. W. Haddock, Beverly; T. Kittredge, Salem; H. W. Mitchell, Hathorne; H. W. Newhall, J. F. O'Shea, J. G. Pinkham, Lynn; E. L. Peirson, J. E. Simpson, Salem.

FRANKLIN.—H. G. Stetson, G. B. Twitchell, Greenfield; N. P. Wood, Northfield.

HAMPDEN.—C. A. Allen, Holyoke; R. P. M. Ames, T. S. Bacon, L. S. Brooks, F. W. Chapin, H. C. Emerson, Springfield; R. E. Dickson, Holyoke; G. E. Fuller, C. W. Jackson, Monson; D. E. Keefe, H. C. Martin, L. A. Prefontaine, A. O. Squier, Springfield; J. P. Schneider, Palmer.

HAMPSHIRE.—J. M. Fay, C. R. Gardner, J. S. Hitchcock, Northampton; W. P. Stutson, Cummington; E. D. Williams, Easthampton.

MIDDLESEX EAST.—A. H. Cowdrey, Stoneham; M. A. Cummings, Winchester; C. Dutton, Wakefield; R. T. Edes, Reading; G. N. P. Mead, Winchester.

MIDDLESEX NORTH.—B. R. Benner, C. M. Frye, A. E. Gillard, T. F. Harrington, W. A. Johnson, W. P. Lawler, G. H. A. Leahey, J. V. Meigs, J. B. O'Connor, C. E. Simpson, Lowell.

MIDDLESEX SOUTH.—E. H. Bigelow, Framingham; T. Chamberlin, Concord; N. F. Chandler, Medford; J. M. Crocker, Cambridge; T. M. Durell, Somerville; G. W. Gay, Chestnut Hill; W. Ela, J. L. Hildreth, Cambridge; J. L. Harriman, Hudson; N. C. B. Haviland, Holliston; A. A. Jackson, Everett, J. A. McDonald, Charlestown; H. B. McIntire, Cambridge; H. E. Marion, Brighton; G. A. Miles, Somerville; J. T. G. Nichols, Cambridge; C. E. Prior, G. Rider, Malden; C. D. Sawin, Charlestown; F. M. Sherman, L. R. Stone, F. R. Stubbs, Newton; F. G. Smith, Somerville; J. E. Somers, E. H. Stevens, F. W. Taylor, H. P. Walcott, Cambridge; W. H. Sylvester, Natick; J. Tolman, Arlington; G. T. Tuttle, Waverley; R. Willis, Somerville; H. A. Wood, A. Worcester, Waltham.

NORFOLK.—B. H. Calkins, Jamaica Plain; S. Crowell, Dorchester; F. H. Davenport, F. W. Draper, Brookline; C. G. Dewey, D. D. Gilbert, Dorchester; H. R. Hitchcock, Hyde Park; C. D. Knowlton, Roxbury; D. Graham, R. W. Hastings, Brookline; W. C. Kite, Milton; J. S. H. Leard, Jamaica Plain; F. D. Lyon, A. A. Macdonald, E. G. Morse, Roxbury; H. T. Mansfield,

Needham; W. Noyes, Dorchester; W. T. Patch, Roxbury; A. P. Perry, T. Smith, Jamaica Plain; L. A. Roberts, Dorchester; E. P. Starbird, Roxbury; H. R. Stedman, Brookline; S. E. Stevens, West Roxbury; W. N. Tenney, Canton; E. Thayer, W. A. White, Roxbury; A. M. Worthington, Dedham; E. C. Wylie, Dorchester.

NORFOLK SOUTH.—O. H. Howe, Cohasset; N. S. Hunting, F. E. Jones, Quincy; E. N. Mayberry, South Weymouth.

PLYMOUTH.—J. H. Averill, Campello; H. W. Dudley, Abington; N. K. Noyes, Duxbury; G. Osgood, Rockland; A. E. Paine, Brockton; F. G. Wheatley, North Abington.

SUFFOLK.—J. B. Ayer, E. G. Brackett, E. H. Bradford, E. M. Buckingham, *Treasurer*, H. L. Burrell, J. F. Bush, A. T. Cabot, *President*, D. W. Cheever, A. Coolidge, Jr., F. J. Cotton, E. G. Cutler, H. Derby, R. H. Fitz, C. F. Folsom, W. W. Gannett, C. M. Green, F. B. Harrington, H. Jackson, B. J. Jeffries, G. F. Jelly, E. P. Joslin, F. I. Knight, A. L. Mason, J. H. McCollom, A. B. Morong, J. G. Mumford, J. C. Munro, A. H. Nichols, C. B. Porter, A. Post, J. J. Putnam, W. L. Richardson, T. M. Rotch, G. H. M. Rowe, F. C. Shattuck, G. B. Shattuck, A. K. Stone, J. B. Swift, H. F. Vickery, O. F. Wadsworth, J. C. Warren, G. H. Washburn, J. C. White, H. Williams, G. Wolcott, Boston; E. S. Boland, M. F. Gavin, South Boston; J. M. Putnam, Chelsea.

WORCESTER.—W. P. Bowers, Clinton; P. P. Comey, W. Davis, J. T. Duggan, O. H. Everett, G. E. Francis, T. H. Gage, A. C. Getchell, D. Harrower, Worcester; E. B. Harvey, Westboro'; S. P. Holbrook, East Douglas; J. O. Marble, H. M. Quinby, Worcester; E. W. Norwood, Spencer; W. G. Reed, Southbridge; E. V. Scribner, L. Wheeler, A. Wood, S. B. Woodward, Worcester.

WORCESTER NORTH.—C. E. Bigelow, Leominster; S. E. Greenwood, Templeton; E. P. Miller, C. H. Rice, F. H. Thompson, *Vice-President*, Fitchburg; E. A. Sawyer, Gardner.

Censors.

BARNSTABLE.—S. T. Davis, Orleans; R. H. Faunce, Sandwich; S. F. Haskins, Cotuit; J. H. Higgins, Marston's Mills; E. M. Parker, *Supervisor*, South Yarmouth.

BERKSHIRE.—H. Bushnell, North Adams; H. Colt, W. W. Leavitt, *Supervisor*, W. M. Mercer, Pittsfield; W. W. Scofield, Dalton.

BRISTOL NORTH.—H. B. Baker, Dighton; F. A. Hubbard, J. B. Murphy, M. Perry, S. D. Presbrey, *Supervisor*, Taunton.

BRISTOL SOUTH.—S. W. Bowen, A. I. Connell, W. A. Dolan, *Supervisor*, Fall River; E. W. Dehu, A. M. Pierce, New Bedford.

ESSEX NORTH.—C. C. Day, Newburyport; C. E. Durant, *Supervisor*, Haverhill; J. A. FitzHugh, Amesbury; F. E. Sweetsir, Merrimac; L. J. Young, Haverhill.

ESSEX SOUTH.—F. W. Baldwin, Danvers; G. H. Gray, Lynn; H. J. Hall, Marblehead; J. H. Knowles, Gloucester; J. E. Simpson, *Supervisor*, Salem.

FRANKLIN.—E. G. Best, Turner's Falls; J. W. Cram, Colrain; G. R. Fessenden, Ashfield; C. G. Trow, Sunderland; N. P. Wood, *Supervisor*, Northfield.

HAMPDEN.—R. P. M. Ames, *Supervisor*, H. W. Van Allen, W. R. Weiser, G. D. Weston, Springfield; D. F. Donoghue, Holyoke.

HAMPSHIRE.—J. J. Burke, Easthampton; I. B. Hayes, Florence; J. S. Hitchcock, *Supervisor*, J. A. Houston, Northampton; D. M. Ryan, Ware.

MIDDLESEX EAST.—M. A. Cummings, *Supervisor*, Winchester; E. S. Jack, Melrose; W. H. Keleher, Woburn; F. E. Park, Stoneham; E. D. Richmond, Reading.

MIDDLESEX NORTH.—R. E. Bell, S. J. Johnson, T. G. McGannon, F. E. Pinkham, C. E. Simpson, *Supervisor*, Lowell.

MIDDLESEX SOUTH.—G. A. Bancroft, South Natick; L. M. Palmer, South Framingham; W. Preble, Cambridge; H. S. Rowen, Brighton; J. Tolman, *Supervisor*, Arlington.

NORFOLK.—F. P. Emerson, Roxbury; G. H. Francis, G. W. Kaan, Brookline; H. R. Hitchcock, *Supervisor*, Hyde Park; E. T. Twitchell, Dorchester.

NORFOLK SOUTH.—C. S. Adams, Wollaston; G. D. Bullock, Weymouth; H. L. Dearing, Braintree; R. McLellan, Quincy; E. N. Mayberry, *Supervisor*, South Weymouth.

PLYMOUTH.—J. E. Bacon, E. A. Chase, Brockton; N. C. King, Campello; A. A. MacKeen, Whitman; G. Osgood, *Supervisor*, Rockland.

SUFFOLK.—F. G. Balch, J. N. Coolidge, C. H. Hare, H. A. Lothrop, A. K. Stone, *Supervisor*, Boston.

WORCESTER.—F. H. Baker, W. T. Clark, M. F. Fallon, E. V. Scribner, *Supervisor*, Worcester; F. H. Clapp, North Grafton.

WORCESTER NORTH.—J. B. Donnelly, West Gardner; E. P. Miller, *Supervisor*, C. W. Spring, Fitchburg; A. E. Mossman, Westminster; A. H. Pierce, Leominster.

Commissioners of Trials.

BARNSTABLE	J. H. Higgins	Marston's Mills.
BERKSHIRE	A. T. Wakefield	Sheffield.
BRISTOL NORTH	E. Washburn	Taunton.
BRISTOL SOUTH	H. S. Swan	Middleboro'.
ESSEX NORTH	J. F. Young	Newburyport.
ESSEX SOUTH	J. Allen	Topsfield.
FRANKLIN	J. D. Seymour	Whately.
HAMPDEN	W. C. Leary	Springfield.
HAMPSHIRE	A. G. Blodgett	Ware.
MIDDLESEX EAST	D. T. Buzzell	Wilmington.
MIDDLESEX NORTH	G. A. Harlow	Tyngsboro'.
MIDDLESEX SOUTH	J. F. Frisbie	Newton.
NORFOLK	E. N. Libby	Roxbury.
NORFOLK SOUTH	G. W. Tinkham	Weymouth.
PLYMOUTH	C. A. Drew	State Farm.
SUFFOLK	M. F. Gavin	Boston.
WORCESTER	C. H. Perry	Worcester.
WORCESTER NORTH	L. G. Chandler	Townsend.

Officers of the District Medical Societies.

BARNSTABLE.—I. O. Cummings, Brewster, *President;* C. E. Harris, Hyannis, *Vice-President;* H. B. Hart, East Dennis, *Secretary;* C. W. Milliken, Barnstable, *Treasurer.*

BERKSHIRE.—J. J. Flynn, Pittsfield, *President;* A. B. Withington, Pittsfield, *Vice-President;* I. S. F. Dodd, Pittsfield, *Secretary;* W. L. Paddock, Pittsfield, *Treasurer;* W. W. Leavitt, *Librarian.*

BRISTOL NORTH.—T. J. Robinson, Taunton, *President;* E. Washburn, Taunton, *Vice-President;* R. D. Dean, Taunton, *Secretary;* W. Y. Fox, Taunton, *Treasurer;* A. R. Crandell, Taunton, *Librarian.*

BRISTOL SOUTH.—C. A. Pratt, New Bedford, *President;* J. H. Gifford, Fall River, *Vice-President;* A. J. Abbe, Fall River, *Secretary and Treasurer.*

ESSEX NORTH.—H. G. Leslie, Amesbury, *President;* W. J. Sullivan, Lawrence, *Vice-President;* M. D. Clarke, Haverhill, *Secretary and Treasurer.*

ESSEX SOUTH.—C. H. Bangs, Lynn, *President;* G. G. Bailey, Ipswich, *Vice-President;* H. E. Sears, Beverly, *Secretary;* G. Z. Goodell, Salem, *Treasurer;* G. K. Blair, Salem, *Librarian.*

FRANKLIN.—C. C. Messer, Turner's Falls, *President;* B. P. Croft, Greenfield, *Vice-President;* C. F. Canady, Greenfield, *Secretary and Treasurer.*

HAMPDEN.—W. M. E. Mellen, Chicopee, *President;* E. H. Guild, Springfield, *Vice-President;* F. J. Ward, Springfield, *Secretary and Treasurer.*

HAMPSHIRE.—C. F. Branch, Amherst, *President;* H. B. Perry, Northampton, *Vice-President;* A. G. Minshall, Northampton, *Secretary;* A. H. Hoadley, Northampton, *Treasurer;* E. W. Brown, Northampton, *Librarian.*

MIDDLESEX EAST.—S. H. Parks, Reading, *President;* H. G. Blake, Woburn, *Vice-President;* C. W. Harlow, Melrose Highlands, *Secretary;* C. Dutton, Wakefield, *Treasurer.*

MIDDLESEX NORTH.—A. Howard, Chelmsford, *President;* W. J. Sleeper, Westford, *Vice-President;* R. J. Meigs, Lowell, *Secretary;* T. B. Smith, Lowell, *Treasurer;* D. N. Patterson, Lowell, *Librarian.*

MIDDLESEX SOUTH.—A. Worcester, Waltham, *President;* T. M. Durell, Somerville, *Vice-President;* F. R. Jouett, Cambridge, *Secretary;* W. Ela, Cambridge, *Treasurer;* E. S. Dow, Allston, *Librarian.*

NORFOLK.—G. W. Clement, Roxbury, *President;* J. A. Tanner, Dorchester, *Vice-President;* J. C. D. Pigeon, Roxbury, *Secretary and Librarian;* E. G. Morse, Roxbury, *Treasurer.*

NORFOLK SOUTH.—J. F. Welch, Quincy, *President;* N. S. Hunting, Quincy, *Vice-President;* S. W. Ellsworth, Quincy, *Secretary, Treasurer and Librarian.*

PLYMOUTH.—W. P. Chisholm, Brockton, *President;* F. G. Wheatley, North Abington, *Vice-President;* A. C. Smith, Brockton, *Secretary and Treasurer;* C. E. Lovell, Whitman, *Librarian.*

SUFFOLK.—Abner Post, Boston, *President;* C. P. Putnam, Boston, *Vice-President;* L. R. G. Crandon, Boston, *Secretary;* W. H. Prescott, Boston, *Treasurer;* B. J. Jeffries, Boston, *Librarian.*

WORCESTER.—E. W. Norwood, Spencer, *President;* D. Harrower, Worcester, *Vice-President;* G. E. Emery, Worcester, *Secretary;* G. O. Ward, Worcester, *Treasurer;* A. C. Getchell, Worcester, *Librarian.*

WORCESTER NORTH.—G. B. Underwood, West Gardner, *President;* A. P. Mason, Fitchburg, *Vice-President;* W. F. Sawyer, Fitchburg, *Secretary;* E. L. Fiske, Fitchburg, *Treasurer;* A. P. Mason, Fitchburg, *Librarian.*

The Massachusetts Medical Society.

PROCEEDINGS OF THE COUNCILLORS.

OCTOBER 4, 1905.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, October 4, 1905, at 12, noon.

The Vice-President, Dr. FRED'K H. THOMPSON, in the chair.

The following Councillors were present:

Berkshire.
J. F. A. Adams.

Bristol North.
S. D. Presbrey.

Bristol South.
A. W. Buck,
W. A. Dolan,
G. deN. Hough,
H. G. Wilbur.

Essex North.
E. H. Noyes,
W. W. Pillsbury,
R. B. Root.

Essex South.
C. M. Cobb,
D. Coggin,
H. K. Foster.

Franklin.
H. G. Stetson,
G. P. Twitchell.

Hampden.
T. S. Bacon,
J. P. Schneider,
A. O. Squier.

Hampshire.
J. S. Hitchcock,
E. D. Williams.

Middlesex East.
A. H. Cowdrey,
C. Dutton,
R. T. Edes,
G. N. P. Mead.

Middlesex North.
C. M. Frye,
T. F. Harrington,
W. A. Johnson,
W. P. Lawler,
G. H. A. Leahey,
J. V. Meigs,
J. B. O'Connor.

Middlesex South.
E. H. Bigelow,
W. Ela,
G. W. Gay,
J. L. Hildreth,
A. A. Jackson,
H. B. McIntire,
H. E. Marion,
G. A. Miles,
J. T. G. Nichols,
C. E. Prior,
G. Ryder,
F. M. Sherman,
F. G. Smith,
J. E. Somers,
E. H. Stevens,
L. R. Stone,
F. R. Stubbs,
W. H. Sylvester
F. W. Taylor,
J. Tolman,
R. Willis.

Norfolk.
D. Graham,
R. W. Hastings.
H. R. Hitchcock,
J. H. S. Leard,
A. A. Macdonald,
H. T. Mansfield,
E. G. Morse,
W. Noyes,
W. T. Patch,
A. P. Perry,
T. Smith,
E. P. Starbird,
S. E. Stevens,
E. Thayer,
W. A. White,
A. M. Worthington,
E. C. Wylie.

Norfolk South.
N. S. Hunting.

Plymouth.
H. W. Dudley,
G. Osgood,
F. G. Wheatley.

Suffolk.
E. S. Boland,
E. M. Buckingham,
H. L. Burrell,
J. F. Bush,
D. W. Cheever,
R. H. Fitz,
C. F. Folsom,
F. B. Harrington,
E. P. Joslin,
A. L. Mason,
C. B. Porter,
A. Post,
J. M. Putnam,
W. L. Richardson,
F. C. Shattuck,
G. B. Shattuck,
A. K. Stone,
J. B. Swift,
O. F. Wadsworth,
H. Williams,
G. Wolcott.

Worcester.
O. H. Everett,
G. E. Francis,
S. P. Holbrook,
J. O. Marble,
E. W. Norwood,
L. Wheeler.

Worcester North.
C. E. Bigelow,
C. H. Rice,
E. A. Sawyer,
F. H. Thompson.
Total, 103.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

Vermont.—C. L. Scudder, of Boston; W. F. Sawyer, of Fitchburg.

New York.—L. A. Jones, of North Adams; C. F. Branch, of Amherst.

New York State Medical Association.—W. W. Gannett, of Boston; F. W. Chapin, of Springfield.

The Committee on Membership and Finance reported through Dr. Stone. In accordance with their recommendation, it was voted that the resignations of the following be accepted:

John B. Beebe, of Great Barrington.
Henry A. Cooke, of Providence, R. I.
Arthur H. Harrington, of New York, N. Y.
John G. W. Knowlton, of Exeter, N. H.
Herman P. Marshall, of Deming, Wash.
Homer T. Partree, of Eatontown, N. J.
Alexander T. Walker, of West Somerville.
Louis F. Wheatley, of Meriden, Conn.

Also that the following be placed on the retired list:

Seabury W. Bowen, of Fall River.
George S. Stebbins, of Springfield.

Also that the following be deprived of the privileges of membership for non-payment of dues:

Edwin W. Bullock, of Haverhill.
Frank F. Carr.
Mary V. Church, of Topeka, Kansas.
Joseph E. Clark, of New York, N. Y.
Charles F. Cogswell, of Brookline.
Joseph M. Conlin, of Chicopee.
Samuel P. Cottrell, of Newport, R. I.
Thomas J. B. Dillon, of Roxbury.
Edward A. Drummond, of Providence, R. I.
Whitfield O. Dunham, of Sioux Falls, S. D.
George W. Grover, of Sheffield.
Ira C. Guptill, of Northborough.
Revere R. Gurley, of New York, N. Y.
Dixi G. Hoyt, of Providence, R. I.
Anselme E. Houle, of Bennington, Vt.
Rufus P. Hubbard.
Charles Storer Knight, of Woodfords, Me.
Edwin Leonard, Jr., of Jersey City, N. J.
Ernest R. Mackintosh, of Lowell.
Jared H. Miller, of Palmer.
Charles O. Murphy, of New York, N. Y.
George M. Muttart, of East Boston.
Luke B. Peck, U. S. Army.
Eustace P. Reid.
Edwin W. Rich, U. S. Army.
Charles C. Rothfuchs, of Boston.
Oliver M. Sheridan, of Jamaica Plain.
Frank E. Smart, of Lowell.
Warren B. Stone.
Harry G. Wyer, of Kirkwood, Mo.

Voted, That Thomas Delap Smith, of Roxbury, be restored to the privileges of membership.

Adjourned at 12.25 P.M.

FRANCIS W. GOSS,
Recording Secretary.

FEBRUARY 7, 1906.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, February 7, 1906, at 12, noon.

The President, Dr. ARTHUR T. CABOT, in the chair.

The following Councillors were present:

Bristol North.
S. D. Presbrey.

Bristol South.
A. W. Buck,
F. E. Stetson,
H. G. Wilbur.

Essex North.
I. J. Clarke,
J. A. Douglass,
J. G. McAllister,
E. H. Noyes,
W. W. Pillsbury,
G. B. Sargent.

Essex South.
C. M. Cobb,
D. Coggin,
H. K. Foster.

Franklin.
G. P. Twitchell.

Hampden.
F. W. Chapin,
C. W. Jackson,
J. P. Schneider.

Middlesex East.
A. H. Cowdrey,
M. A. Cummings,
C. Dutton,
R. T. Edes.

Middlesex North.
W. A. Johnson,
W. P. Lawler,
G. H. A. Leahey,
J. V. Meigs,
J. B. O'Connor.

Middlesex South.
E. H. Bigelow,
J. M. Crocker,
W. Ela,
G. W. Gay,
J. L. Hildreth,
J. A. McDonald,
H. B. McIntire,
H. E. Marion,
G. Ryder,
F. M. Sherman,
E. H. Stevens,
L. R. Stone,
F. R. Stubbs,
W. H. Sylvester,
F. W. Taylor,
J. Tolman,
H. P. Walcott,
R. Willis,
A. Worcester.

Norfolk.
D. D. Gilbert,
D. Graham,
R. W. Hastings,
W. C. Kite,
F. D. Lyon,
E. G. Morse,
W. Noyes,
W. T. Patch,
A. P. Perry,
T. Smith,
E. P. Starbird,
S. E. Stevens,
E. Thayer,
W. A. White,
A. M. Worthington,
E. C. Wylie.

Norfolk South.
O. H. Howe,
E. N. Mayberry.

Plymouth.
J. H. Averill,
G. Osgood,
A. E. Paine,
F. G. Wheatley.

Suffolk.
J. B. Ayer,
E. S. Boland,
E. M. Buckingham,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
A. Coolidge, Jr.,
E. G. Cutler,
C. F. Folsom,
C. M. Green,
F. B. Harrington,
E. P. Joslin,
J. G. Mumford,
J. C. Munro,

A. H. Nichols,
C. B. Porter,
A. Post,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
F. C. Shattuck,
G. B. Shattuck,
A. K. Stone,
J. B. Swift,
H. F. Vickery,
O. F. Wadsworth,
H. Williams,
G. Wolcott.

Worcester.

P. P. Comey,
W. Davis,
O. H. Everett,
G. E. Francis,
D. Harrower,
E. B. Harvey,
S. P. Holbrook,
J. O. Marble,
S. B. Woodward.

Worcester North.

C. E. Bigelow,
F. H. Thompson.

Total, 106.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other Medical Societies:

American Medical Association, 1906 and 1907.—E. H. Bradford, of Boston; W. P. Bowers, of Clinton; F. G. Wheatley, of North Abington.

Alternates.—G. W. Gay, of Newton; Homer Gage, of Worcester; Charles Harrington, of Boston.

Maine.—F. W. Anthony, of Bradford; C. E. Prior, of Malden.

New Hampshire.—T. F. Harrington, of Lowell; H. K. Faulkner, of Keene, N. H.

Rhode Island.—F. B. Lund, of Boston; J. C. Pegram, Jr., of Providence, R. I.

Connecticut.—R. H. Seelye, of Springfield; O. F. Rogers, of Dorchester.

New Jersey.—A. H. Hodgdon, of Dedham; H. A. Lothrop, of Boston.

The following Committee was appointed:

To Audit the Treasurer's Accounts.—M. P. Smithwick, E. A. Locke.

The Committee on Membership and Finance reported through Dr. Stone. In accordance with their recommendation it was voted that the resignations of the following be accepted:

Charles Burleigh, of Malden.
Frederick B. Willard, of Hartford, Conn.

Also that the following be placed on the retired list:

Chester M. Barton, of Hatfield.
George A. Bragdon, of Middletown, Conn.
Nomus Paige, of Taunton.
N. Morton Ransom, of Somerville.

Also that the following be deprived of the privileges of membership for non-payment of dues:

Bradford Allen, of Nashua, N. H.
Theodore C. Burnett, of San Francisco, Cal.
Francis X. Crawford, of Boston.
Benedict Donovan, of Brockton.
George F. Garland, of Waltham.
Rufus H. Hall, of Everett.
Charles C. Holcombe, of Lee.
George C. Littlefield, of Salem.
Philip W. T. Moxom, of Flatbush, N. Y.
Frank H. Parker, of Malden.
Harry M. Peggs, of Somerville.
Winfred C. Pike, of Dorchester.
Harris S. Pomeroy, of Peabody.

Dr. F. C. Shattuck moved, that a Committee of five be appointed by the President to consider and report on the advisability of such changes in the By-Laws as may bring them into conformity with present-day practice.

After remarks by Drs. Worcester and Sherman it was voted, that Dr. Shattuck's motion be adopted.

[The President subsequently appointed Drs. F. C. Shattuck, W. P. Bowers, F. G. Wheatley, E. H. Stevens and F. W. Chapin to constitute the Committee.]

The President stated that owing to the meeting of the American Medical Association in the week previous to the next Annual Meeting of the Society, it seemed advisable to the Committees To Procure Scientific Papers and Of Arrangements to omit the meetings of the Sections on the latter occasion.

Voted, To adopt the recommendation.

Adjourned at 1.05 P.M.

FRANCIS W. GOSS,
Recording Secretary.

JUNE 12, 1906.

THE ANNNAL MEETING of the Councillors was held in the Medical Library, Boston, on Tuesday, June 12, 1906, at 5 P.M.

The President, Dr. ARTHUR T. CABOT, in the chair.

The following Councillors were present:

Berkshire.
J. F. A. Adams,
O. J. Brown,
H. Bushnell,
I. S. F. Dodd.

Bristol North.
A. S. Deane,
W. Y. Fox,
S. D. Presbrey.

Bristol South.
A. W. Buck,
W. A. Dolan,
H. G. Wilbur.

Essex North.
J. F. Burnham,
I. J. Clarke,
J. A. Hogan,
E. H. Noyes,
W. W. Pillsbury,
L. J. Young.

Essex South.
T. Dwight,
W. W. Eaton,
T. Kittredge,
H. E. Sears,
J. Shanahan,
F. E. Stone,
A. M. Tupper.

Franklin.
H. G. Stetson,
G. P. Twitchell.

Hampden.
E. A. Bates,
C. W. Jackson,
J. P. Schneider.

Hampshire.
A. G. Blodgett,
F. C. Bruce,
H. T. Shores.

Middlesex East.
A. H. Cowdrey,
R. T. Edes,
G. N. P. Mead.

Middlesex North.
W. P. Lawler,
J. H. Nichols,
C. E. Simpson.

Middlesex South.
N. F. Chandler,
J. M. Crocker,
W. Ela,
D. S. Flewelling,
G. W. Gay,
J. L. Hildreth,
A. A. Jackson,
J. A.McDonald,
H. E. Marion,
J. A. Mead,
G. A. Miles,
J. T. G. Nichols,
W. Preble,
H. S. Rowen,
F. M. Sherman,
E. H. Stevens,
L. R. Stone,
I. E. Stowe,
W. H. Sylvester,
F. W. Taylor,
J. Tolman,
H. P. Walcott,
A. M. Wilkins,
H. A. Wood.

Norfolk.
D. N. Blakely,
B. H. Calkins,
H. L. Chase,
S. W. Crittenden,
G. H. Francis,
D. D. Gilbert,
D. Graham,
J. S. Greene,
W. C. Kite,
C. D. Knowlton,
A. A. Macdonald,
H. T. Mansfield,
M. L. Marr,
E. G. Morse,
M. V. Pierce,
O. F. Rogers,
W. H. Robinson,
T. Smith,
H. B. Stevens,
S. E. Stevens,
E. Thayer,
W. A. White,
A. M. Worthington.

Norfolk South.
N. S. Hunting,
E. N. Mayberry.

Plymouth.
H. W. Dudley,
G. Osgood,
A. E. Paine,
F. G. Wheatley.

Suffolk.
H. D. Arnold,
J. B. Ayer,
E. S. Boland,
E. G. Brackett,
E. H. Bradford,
E. M. Buckingham,
H. L. Burrell,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
A. Coolidge, Jr.,
F. J. Cotton,
E. G. Cutler,
H. Derby,
R. H. Fitz,
C. F. Folsom,
C. M. Green,
F. B. Harrington,
B. J. Jeffries,
G. F. Jelly,
F. I. Knight,
A. B. Morong,
J. G. Mumford,
J. C. Munro,
A. H. Nichols,
C. B. Porter,
A. Post,
C. P. Putnam,
J. M. Putnam,
W. L. Richardson,
G. G. Sears,
F. C. Shattuck,
A. K. Stone,
H. F. Vickery,
O. F. Wadsworth,
J. C. Warren,
H. Williams,
G. Wolcott.

Worcester.
W. P. Bowers,
O. H. Everett,
H. Gage,
A. C. Getchell,
R. W. Greene,
D. Harrower,
E. B. Harvey,
S. P. Holbrook,
A. Wood,
S. B. Woodward.

Worcester North.
C. E. Bigelow,
H. H. Lyons,
E. P. Miller,
E. A. Sawyer,
F. H. Thompson.
Total, 143.

The record of the last meeting was read and accepted.

The Secretary reported that 283 Fellows had been admitted since the last annual meeting, and that 41 deaths had been recorded.

The Treasurer, Dr. BUCKINGHAM, read his annual report.

The Auditing Committee reported that they found the accounts correctly cast and properly vouched, and that the Treasurer has in his possession the securities called for.

Voted, To accept the Treasurer's report.

The Committee on Membership and Finance reported through Dr. Stone, and recommended that $4,500.00 of the surplus in the treasury be distributed among the District Societies.

Voted, That the recommendation be adopted.

In accordance with the recommendation of the same Committee it was voted, that the resignations of the following be accepted:

Annie M. Blossom, of North Adams.
Zelly A. Bonoff, of New Haven, Conn.
Mary J. Brewster, of Northampton.
M. Augusta Camp, of Northampton.

Elliott M. Clarke, of Denver, Colo.
Margaret E. V. Fraser, of Denver, Colo.
Claude P. Jones, of Vineland, N. J.
Charles S. Little, of Laconia, N. H.
Jay Perkins, of Providence, R. I.
Charles G. Stearns, of East Dennis.

Also that the following be placed on the retired list:

Cornelius A. Ahearne, of Lynn.
John A. Douglass, of Amesbury.
Isaac H. Hazelton, of Wellesley Hills.
Francis A. Howe, of Newburyport.
William W. Leavitt, of Pittsfield.
George E. Pinkham, of Lowell.
Albert R. Rice, of Springfield.
Isaac W. Starbird, of Roxbury.

The Committee On Publications through Dr. Wadsworth presented their report.

Dr. Sears for the Committee on State and National Legislation reported that

During the present session of the Legislature they have appeared either personally or by representatives before the committees of the State Legislature in favor of the following bills:

To regulate the height of buildings outside of the city of Boston.

To provide for the better care of the health of pupils in public schools.

To regulate the sale of patent, proprietary, and other medicines containing poisonous drugs.

To regulate the sale of adulterated foods and drugs.

According to the last bulletin issued by the Legislature, the only one of these bills which has been enacted is that regulating the sale of adulterated foods and drugs. The others have passed one branch of the Legislature, but have not been reached in the other. We have not succeeded in all cases in obtaining as stringent legislation as was desired.

Opposition to the bill annually introduced to prohibit vivisection has been left as usual in the hands of Dr. H. C. Ernst. The bill was defeated in committee.

The hardest work of this committee was done in opposing a bill to establish a State Board of Examination and Registration

in Osteopathy. It was finally killed in the Legislature itself as a result in great measure of the work done by the Auxiliary Legislative Committee of the Society.

This committee believes that the Auxiliary Legislative Committee is of great use in directing popular opinion and in bringing proper pressure to bear upon the members of the General Court, and that a larger committee, in which each senatorial district shall be represented by a member, would be a more useful body than the present one in which each district of the Society is represented.

Voted, To accept the report of the Committee and to adopt its recommendation regarding the Auxiliary Legislative Committee.

The Librarian, Dr. Brigham, presented his report.

Voted, That vacancies in Boards of Censors be filled as follows:
In Essex North by W. H. Merrill, of Lawrence.
In Norfolk South by V. M. Tirrell, of South Weymouth.

The Committee on Nominations reported a list of candidates for the offices of the Society for the ensuing year, and the same were elected by ballot:

President . . GEORGE W. GAY, of Newton.
Vice-President . LEONARD WHEELER, of Worcester.
Treasurer . EDWARD M. BUCKINGHAM, of Boston.
Corresponding Sec'y CHARLES W. SWAN, of Lowell.
Recording Secretary FRANCIS W. GOSS, of Roxbury.
Librarian . EDWIN H. BRIGHAM, of Brookline.

J. F. ALLEYN ADAMS, of Pittsfield, was chosed Orator for the annual meeting of the Society in 1907.

Voted, That the next annual meeting of the Society be held in Boston on the second Wednesday in June, 1907.

The following Standing Committees were appointed:

Of Arrangements.

A. P. Perry, L. W. Gilbert,
G. S. C. Badger, C. C. Simmons,
Lincoln Davis, G. H. Francis.

On Publications.

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

On Membership and Finance.

F. W. Goss, Walter Ela, C. M. Green,
A. Coolidge, Jr., H. A. Wood.

To Procure Scientific Papers.

J. B. Blake, B. P. Croft, R. C. Larrabee,
M. D. Clarke, Charles Harrington.

On Ethics and Discipline.

C. G. Carleton, Edward Cowles, J. F. A. Adams,
Leonard Wheeler, J. A. Gage.

On Medical Diplomas.

H. E. Marion, O. F. Rogers, H. W. Newhall.

On State and National Legislation.

G. W. Gay, H. P. Bowditch, S. D. Presbrey,
G. G. Sears, D. D. Gilbert.

The resignation of Dr. Charles W. Swan as Corresponding Secretary was presented.

Voted, To accept the resignation.

Dr. Cheever spoke of the prolonged service of Dr. Swan as an Officer of the Society and moved that the sincere thanks and sympathy of the Councillors be presented to Dr. Swan. Adopted.

The Recording Secretary, Dr. Goss, was elected also Corresponding Secretary for the ensuing year.

Dr. F. C. Shattuck, for the Committee appointed at the last meeting to consider and report on the advisability of such changes in the By-Laws as may bring them into conformity with present day practice, reported recommending the following changes:

I. In By-Law I., line 9, omit "and surgical."
Omit lines 9 to 13 inclusive, "and"—"years."
In line 15 substitute "treat" for "cure."
In lines 16 and 17 omit "spiritualism, homœopathy, allopathy, Thomsonianism, eclecticism or"—and "other irregular or."

If the proposed changes are adopted, the By-Law would read:

Candidates for admission into the Massachusetts Medical Society may be either male or female; and every candidate must by

examination, as hereinafter provided, satisfy the Censors of the Society that he is not less than twenty-one years of age; that he is of sound mind, and good moral character; that he has a good general English education; that he has a knowledge of the principles of experimental philosophy; that he has such an acquaintance with the Latin language as is necessary for a good medical education; that he has studied medicine at an authorized medical school, recognized by the Councillors of the Society, and has received a diploma from such School; that he does not profess to treat diseases by, nor intend to practise, any exclusive system, generally recognized as such by the profession or declared so by the Councillors of the Society; and that he possesses such other qualifications as the Society may deem necessary.

II. Omit By-Law IX. in toto.

III. Change By-Law X. so as to read:

Any Fellow of this Society who advertises for sale, or otherwise offers to the public, any medicine the composition of which he keeps secret, or who offers to treat any disease by any such secret medicine, or by any secret treatment, shall be liable to expulsion, or to such other penalty as a Board of Trial may have power to inflict.

Dr. Cheever moved to amend the Committee's report by inserting in By-Law I., line 16, after the word "practise" the words "nor to assume the name of," so that the clause would read "that he does not profess to treat diseases by, nor intend to practise, nor to assume the name of, any exclusive system," etc.

Discussion but no action followed the offering of these resolutions, and they were reserved in accordance with the By-Laws to be reported to the Society at its meeting to-morrow.

Adjourned at 6.20 P.M.

FRANCIS W. GOSS,
Recording Secretary.

The Massachusetts Medical Society.

PROCEEDINGS OF THE SOCIETY.

ANNUAL MEETING.

First Day.

June 12, 1906.

The new buildings of the Harvard Medical School, Boston, were open to members of the Society during the afternoon.

There was an exhibit of specimens and appáratus in the museum in the Administration Building and in the Building of Anatomy.

From 3 to 5 P.M. a series of demonstrations under the direction of Dr. W. T. Councilman was given.

At 8 P.M., in the Medical Library, the Shattuck Lecture was delivered by Victor C. Vaughan, M.D., of Ann Arbor, Mich.

Following the lecture a social hour was passed.

Second Day.

June 13, 1906.

The Society met in Mechanic Building, Boston, on Wednesday, June 13, 1906, at 9 A.M., for the exercises of the one hundred and twenty-fifth Anniversary.

The President, Dr. Arthur T. Cabot, in the chair.

The record of the last annual meeting was read and accepted.

The Secretary announced that 283 Fellows had been admitted during the past year and that 41 deaths had been recorded.

The following are the lists:

Admissions reported from June 13, 1905, to June 12, 1906.

1906	Adams, Charles Waldron	Cambridge.
1906	Arkin, Louis	Roxbury.
1905	Arms, Burdett Loomis	Brookline.
1905	Ascher, Joseph	Boston.
1905	Atwater, James Billings	Westfield.
1906	Austin, Albert Elmer	Medway.
1906	Balboni, Gerardo Monari	Boston.
1905	Bardwell, Frederick Albert	Roxbury.
1906	Barnes, William Lester	Lexington.
1905	Bartlett, Walter Oscar	Roxbury.
1906	Bartol, Edward Francis Washburn	Milton.
1906	Beach, Sylvester Judd	Boston.
1905	Beckett, Francis Henry	Fall River.
1906	Bennett, Hamlin Perley	Woodstock, Vt.
1905	Bianco, Joseph Anthony	East Boston.
1906	Bigelow, Alice Houghton	Jamaica Plain.
1905	Bigelow, Edward Bridge	Worcester.
1906	Blake, Gerald	Boston.
1906	Boardman, William Parsons	Boston.
1906	Borden, George Edward	Fall River.
1906	Bosworth, Freeman Dodd	Tewksbury.
1906	Brady, Frank Robert	Lowell.
1905	Brennan, Thomas Joseph	New Bedford.
1906	Brindisi, Rocco	Boston.
1906	Brooks Edith May	Roxbury.
1905	Brown, Edward Manning	Springfield.
1906	Brown, Wallace Everett	North Adams.
1906	Bryant, Clarence Edmund	Cambridge.
1906	Buckley, William Stephen	Brighton.
1906	Burnett, Francis Lowell	Cambridge.
1906	Bushnell, Homer Andrew	North Adams.
1905	Butler, Joel Ives	Springfield.
1906	Carney, Patrick Joseph	Dorchester.
1906	Carr, Percy Whitman	Hyde Park.
1905	Carroll, John Joseph	Holyoke.
1905	Carvill, Lizzie Maud	Somerville.
1905	Cavanaugh, Thomas Edward	Holyoke.
1905	Celce, Frank Frederick	Holyoke.
1905	Celce, Jean Henriette	Holyoke.
1906	Chisholm, Miles Dudley	Westfield.
1906	Churchill, John Darling	Plymouth.
1906	Clark, John Donovan	Auburndale.

1905	Clark, Nelson Henry	Boston.
1905	Cockett, Marguerite Standish	Roxbury.
1905	Coffrey, William Francis	Westfield.
1905	Coleman, David Charles	Beverly.
1905	Collins, Aubrey John	Mattapan.
1906	Cooper, Alden Vernon	Palmer.
1905	Corey, Frederick Hall	Roxbury.
1906	Costello, John Henry	Dorchester.
1905	Coté, Honoré Joseph	Boston.
1905	Cox, Simon Francis	Boston.
1905	Craig, William Gibson	Springfield.
1906	Crane, Bayard Taylor	Rutland.
1905	Cregg, Francis Aloysius	Lowell.
1905	Curley, Clarence Proctor	Provincetown.
1905	Curran, Simon Francis	Dorchester.
1906	Cushing, Arthur Alden	Brookline.
1905	Dacey, Cornelius Joseph	Brockton.
1906	Dana, Harold Ward	Boston.
1906	Danforth, Harland Abbott	Lynn.
1906	Davidson, Henry James Duff	Springfield.
1906	Davis, Charles Augustus	Palmer.
1906	Davis, Minot Flagg	Boston.
1905	Davison, Arthur Howard	Dorchester.
1905	Day, Hilbert Francis	Boston.
1906	Dearborn, Henry Hale	Leominster.
1905	Deering, George Edwin	Worcester.
1906	Denning, Edward John	South Boston.
1906	Dexter, Richard	Boston.
1905	Dore, Francis James	Roxbury.
1905	Douglas, Archibald John	Westfield.
1906	Dubois, Eoline Church	Cambridge.
1906	Duffee, Thomas Edward	Winchendon.
1905	Dugdale, Frederick	Lowell.
1906	Dunbar, Francis Herbert	Boston.
1905	Duryee, Albert Plummer	Brockton.
1906	Dutcher, William Austin	Boston.
1906	Dutton, Julius Maltby	Westfield.
1905	Dwyer, William Joseph	Cambridge.
1906	Eastman, Theodore Jewett	Boston.
1906	Ehrenfried, Albert	Boston.
1906	Eldredge, Harvey Loud	East Boston.
1906	Ellam, Herbert William	Worcester.
1905	Ellis, Edward Keith	Boston.
1905	Emmons, Arthur Brewster	Brookline.
1906	Fabyan, Marshal	Brookline.
1906	Farmer, William Francis	Holden.
1905	Faunce, Calvin Barstow, Jr.	Jamaica Plain.

1905	Faxon, Dora Winifred	Taunton.
1905	Fenwick, George Benson	Worcester.
1906	Field, Henry Martyn	Dorchester.
1906	Fitzgerald, John Joseph	Haverhill.
1905	Flynn, Henry Lawrence	Roxbury.
1905	Furrer, Arnold Frotcham	Cleveland, O.
1905	Gabler, George Lewis	Holyoke.
1905	Gage, Fred Leon	Worcester.
1905	Gates, George Cushman	Chicopee.
1906	Gile, Frank Herbert, Jr.	Melrose Highlands.
1906	Gill, Mary Eva	Dorchester.
1905	Gillis, John Ewen	West Somerville.
1906	Gilman, Charles Sleeper	Boston.
1906	Gilpatrick, Roy Hawkes	Boston.
1906	Glass, James	South Framingham.
1906	Goddard, Samuel Warren	Brockton.
1905	Goddu, Louis Adalore Oliver	Beverly.
1906	Good, Frederick Leo	Boston.
1905	Gorham, George Hartley	Tabernilla, C. Z.
1905	Graves, Benjamin Augustus	Dorchester.
1906	Graves, James Chapman, Jr.	Boston.
1905	Haché, Henry Clement	Somerville.
1906	Halligan, Edward Maurice	South Boston.
1906	Halsall, Mary Elizabeth	East Boston.
1906	Hammond, Charles	Hanover.
1906	Hancock, Albert William	Lawrence.
1905	Hanson, William Clinton	Cambridge.
1906	Hardwick, Sydney Curtis	Quincy.
1905	Harrington, Robert Brine	Northampton.
1906	Harrison, Henry	Boston.
1906	Hartman, Gustave	Lynn.
1906	Hartnett, Edward Daniel	East Boston.
1906	Hatch, Royal	Boston
1906	Heffernan, David Aloysius	Boston.
1906	Hicks, George Henry	Fall River.
1905	Hinchliffe, Frederick	Lowell.
1906	Hoitt, Charles Lewis	Lynn.
1906	Holt, Harry Frye	Worcester.
1905	Hooker, Stuart VanRenseelaer	Boston.
1906	Hopkinson, George	Boston.
1905	Hornbrook, Francis Wheeler	Newton.
1906	Horne, Lester Wallace	Acushnet.
1906	Hosley, Walter Alexis	Boston.
1905	Hunt, Alice Elizabeth Palmer	Holyoke.
1905	Hunter, Norman McLeod	Hudson.
1906	Hurley, Edward Daniel	South Boston.
1906	Janes, Arthur Percy	Melrose Highlands.

1905	Jennings, Curtis Hermon	Fitchburg.
1905	Jones, Frederick Elmer	Roxbury.
1905	Jones, Wellington West	Housatonic.
1905	Jordan, John Franklin	Peabody.
1906	Keeler, William Basil	Roxbury.
1905	Keeley, Ambrose Augustine	Fall River.
1905	Kennedy, Edward Anthony	Monson.
1906	Kidner, Frederick Clinton	Boston.
1906	Kingsbury, Walter Warren	Malden.
1905	Kingsley, Patrick Joseph	Dorchester.
1906	Lahey, Francis Howard	Boston.
1905	Lane, John William	Boston.
1906	Law, Katharine Hayes	Boston.
1905	Leary, Chrysostom John	New Bedford.
1905	Le Gro, Lester Burnside	Haverhill.
1906	Lewis, Elisha Sears	Princeton.
1906	Lewis, James Prince	Boston.
1906	Long, Merritt Allen	Manchester.
1905	Longfellow, Henry Nathan	Roxbury.
1905	Lowell, William Holbrook	Boston.
1905	Lowenstein, Fred Philip	Westfield.
1905	Lucas, Julian Dyer	Roxbury.
1906	McAllester, John Joseph Hector	New Bedford.
1905	McAllester, Ralph William	Everett.
1906	McCarthy, Francis Patrick	Jamaica Plain.
1906	McCarthy, Timothy Francis	East Boston.
1905	McCauley, James Douthard	Cambridge.
1906	McCormick, John Joseph	South Boston.
1906	Macdonald, Frederick Cornelius	Roxbury.
1906	MacKerrow, Horace Gilford	Worcester.
1906	MacOdrum, Angus	Boston.
1905	McSheehy, Theobald Coleman	Worcester.
1905	Magrath, George Burgess	Boston.
1906	Maguire, Eugene Leo	South Boston.
1905	Mahoney, Francis Xavier	Dorchester.
1905	Maloney, Thomas Aloysius	Springfield.
1905	Marshall, Harold Kenneth	Dorchester.
1906	Mather, John Adams	Colrain.
1906	Means, Philip Corydon	Chelsea.
1905	Medalia, Leon Samuel	Boston.
1905	Merrill, Ayres Phillip	Pittsfield.
1906	Miller, Albert Eber	Needham.
1906	Miller, Malcolm Dean	Boston.
1906	Miller, Vesta Delphene	Needham.
1906	Mills, Alvah Vernon	Brookline.
1906	Miner, Harold Edson	Holyoke.
1906	Mitchell, Elmer Wesley	Fall River.

1906	Morrow, William Robert	South Framingham.
1905	Morse, Nathaniel Niles	New York, N. Y.
1905	Morse, Vernon Harcourt Chipman	Cambridge.
1906	Mullin, Estella Loretta	Worcester.
1905	Mullins, Eugene Norton	Baldwinsville.
1906	Munroe, Harrington Bennett	Lynn.
1905	Murphy, Frederick Paul	Lowell.
1905	Murphy, Frederick Vincent	Attleborough.
1905	Nagle, Evelyn Wyman	Boston.
1905	Noyes, Margaret Louise	Boston.
1905	Noyes, William Nelson	Fitchburg.
1906	Oak, Charles Arthur	Revere.
1905	O'Brien, Daniel Paul	New Bedford.
1905	O'Brien, Joseph Jeremiah	Dorchester.
1905	O'Connell, Andrew Edward	Worcester.
1906	O'Connor, John Christopher	Salem.
1906	Oliver, Everard Lawrence	Boston.
1905	O'Malley, Edward Francis	Fitchburg.
1905	Ordway, Mabel Dyer	Boston.
1905	Ordway, Thomas	Boston.
1906	Orton, Samuel Torrey	Boston.
1905	O'Sullivan, Francis Michael Aloysius	Lowell.
1905	Paine, Alonzo Kingman	Boston.
1905	Paquin, Joseph Ubalde	New Bedford.
1906	Parker, Albert Munro	Boston.
1906	Pascoe, William Whelan	Savoy.
1905	Pattrell, Arthur Ellis	North Grafton.
1906	Pease, Lewis Waite	Hyannis.
1906	Potter, Philip Sheridan	North Adams.
1905	Powers, Herbert Hale	Brookline.
1905	Prescott, Henry Dudley	New Bedford.
1906	Purvis, Charles Burleigh	Boston.
1906	Reynolds, John Timothy	Quincy.
1905	Rice, Alexander Hamilton	Boston.
1906	Rice, Allen Galpin	Boston.
1906	Riley, John Henry	North Adams.
1906	Roach, Alfred John	Lowell.
1906	Robbins, Chandler	Boston.
1906	Robbins, William Bradford	Boston.
1906	Roberts, Frederick Albert	Wilmington.
1906	Robinson, David	Boston.
1906	Rogers, Mark Homer	Boston.
1906	Rowe, Carleton Allen	Tewksbury.
1906	Rushford, Edward Allan	Salem.
1905	Russell, Charles Bradley	Boston.
1905	Russell, Walter Burton	Springfield.
1906	Ryder, Delano Richmond	Fall River.

1906	Scannell, James Joseph	Boston.
1906	Schmidt, Richard Diedrich	Dorchester.
1905	Schofield, Otho Lester	Wellesley.
1905	Seaver, Edwin Pliny, Jr.	Waban.
1905	Sedgewick, Otis White	Wales.
1906	Shaw, Henry	Boston.
1906	Shea, Michael Ignatius	Chicopee Falls.
1906	Shohan, Joseph	Roxbury.
1905	Shoninger, Lee Simon	Boston.
1905	Sleeper, Frank Warren	Dorchester.
1906	Smith, Stafford Baker	Haverhill.
1905	Smyth, Patrick Somers	Boston.
1905	Staples, Clarence Hathorne	Malden.
1906	Stevens, Harold Elmer Ellsworth	Boston.
1905	Stickney, Whitman Gibson	Beverly.
1905	Stone, Ralph Edgarton	Beverly.
1905	Storey, Thomas Andrew	Boston.
1906	Storrs, Henry Randolph	Boston.
1905	Streeter, John Frank	Springfield.
1905	Sturnick, Max	Roxbury.
1906	Sullivan, Cornelius Augustine	South Braintree.
1905	Sullivan, Joseph Lawrence	Roxbury.
1905	Sylvester, Albie Warren	Pittsfield.
1906	Talbot, Fritz Bradley	Brookline.
1906	Thompson, George Hocken	North Adams.
1906	Tinkham, Oliver Goldsmith	Weymouth.
1905	Tobey, George Loring, Jr.	Boston.
1906	Tolman, Henry, Jr.	Boston.
1906	Toohey, Thomas Victor	Roxbury.
1905	Tower, Freeman Augustus	Worcester.
1905	Tracy, John Matthew	Springfield.
1906	Trainor, Joseph Aloysius	Cambridge.
1906	Trask, John Wilson	Lynn.
1906	Trayes, William Henry, Jr.	Boston.
1905	Turner, Charles Humphrey	Haverhill.
1906	Turner, William George	Fall River.
1905	Tyler, Winsor Marrett	New York, N. Y.
1906	Tyzzer, Ernest Edward	Wakefield.
1905	Walker, William Dacre	Boston.
1905	Warren, Edward Dane	Holyoke.
1905	Warren, Franklin Lafayette	Bridgewater.
1906	Way-Allen, Eleanor	Walpole.
1906	Whelan, Charles	Boston.
1905	Whitcher, Burr Royce	Boston.
1906	Whitman, Luther Oakes	Melrose Highlands
1905	Whitney, Ray Lester	Ashburnham.

1906	Whittemore, Wyman	Boston.
1906	Wilinsky, Charles Francis	Boston.
1906	Wilkinson, William Macdonald	Lawrence.
1906	Wilson, John Edward	Boston.
1905	Young, Ralph Randal	Roxbury.
1905	Young, Roy Demas	Arlington.
1906	Zimmermann, Louisa Juliet	Worcester.

Total, 283

List of Deceased Fellows.

Admitted.	Name.	Residence.	Date of Death.	Age.
1885	Barnard, Rebecca	Worcester	Sep. 9, 1905	68
1867	Bass, William	Dedham	July 21, 1905	95
1898	Beattie, Robert Fowler	Brookline	Aug. 6, 1905	32
1893	Blake, Warren Perkins	Springfield	Feb. 15, 1906	47
1843	Brooks, Samuel Doolittle	Springfield	Feb. 26, 1906	89
1893	Burrell, Benjamin Henry	Roxbury	April 23, 1906	53
1871	Chadwick, James Read	Boston	Sep. 24, 1905	60
1874	Chase, Eli Ayer	Brockton	Oct. 3, 1905	58
1861	Curtis, Hall	Boston	June 1, 1906	71
1870	Davis, Wesley	Worcester	Feb. 16, 1906	64
1895	Dever, Charles Edward	Lynn	Oct. 24, 1905	43
1846	Doane, George Washington	Hyannis	Dec. 28, 1905	81
1896	French, John Innes	Winchester	Nov. 18, 1905	44
1882	Galligan, Edward Francis	Taunton	Sep. 26, 1905	48
1866	Gardner, William Wallace	Springfield	Dec. 11, 1905	79
1880	Garrigan, Thomas James	No. Brookfield	April 20, 1906	51
1874	Gilfillan, Thomas	Northampton	Sep. 9, 1905	77
1878	Harriman, James Lang	Hudson	Dec. 28, 1905	78
1895	Hedenberg, James	Medford	July 25, 1905	74
1896	Holland, James William	Westfield	Dec. 29, 1905	37
1856	Hyde, George Smith	Boston	Dec. 11, 1905	74
1875	Kingsbury, Joseph Byron	Holbrook	March 24, 1906	71
1898	Lockwood, George Bertrand	Sharon	July 12, 1905	31
1897	Mahon, James Arthur	Dorchester	Sep. 14, 1905	40
1880	Mellen, William Michael Edward	Chicopee	May 14, 1906	58
1891	Moulton, Charles Fred	West Roxbury	April 24, 1906	40
1860	Munsell, George Nelson	Harwich	Nov. 1, 1905	69
1867	Park, John Gray	Groton	Aug. 29, 1905	67
1893	Parker, Charles Edwin	Princeton	July 10, 1905	41
1897	Pegram, John Combe, Jr.	Providence, R.I.	April 26, 1906	35
1899	Pettee, John Harris	Roxbury	March 5, 1906	34
1875	Pierce, Andrew Martin	New Bedford	Nov. 6, 1905	53
1897	Prefontaine, Louis Aurele	Springfield	June 19, 1905	39
1895	Scannell, Michael Edward	Worcester	Nov. 16, 1905	41
1904	Shead, Edward Wadsworth	Cambridge	Aug. 19, 1905	31
1895	Sheahan, Joseph Maurice	Quincy	Sep. 21, 1905	53
1895	Van Rensselaer, Henry Rensselaer	Lanesborough	Feb. 13, 1905	51
1863	Warner, Emerson	Worcester	Dec. 24, 1905	74
1849	Williams, Jacob Lafayette	Boston	May 16, 1906	82
1871	Wood, Edward Stickney	Boston	July 11, 1905	59
1904	Zabriskie, Fred Templeton	Pittsfield	Nov. 5, 1905	33

Total, 41

The Treasurer's report was read.

The record of the Board of Trial in the following case was read.

Voted, That the sentence of the Board of Trial be imposed, and that

Percy DeMille McLeod, of Boston,

be and is expelled from his membership of The Massachusetts Medical Society.

The Secretary reported from the Councillors the following proposed amendments to the By-Laws, as proposed by a Committee appointed to consider and report on the advisability of such changes in the By-Laws as may bring them into conformity with present day practice, recommending the following changes:

I. In By-Law I., line 9, omit "and surgical."
Omit lines 9 to 13 inclusive, "and" — "years."
In line 15 substitute "treat" for "cure."
In lines 16 and 17 omit "spiritualism, homœopathy, allopathy, Thomsonianism, eclecticism or" — and "other irregular or."

Candidates for admission into The Massachusetts Medical Society may be either male or female; and every candidate must by examination, as hereinafter provided, satisfy the Censors of the Society that he is not less than twenty-one years of age; that he is of sound mind, and good moral character; that he has a good general English education; that he has a knowledge of the principles of experimental philosophy; that he has such an acquaintance with the Latin language as is necessary for a good medical education; that he has studied medicine at an authorized medical school, recognized by the Councillors of the Society, and has received a diploma from such School; that he does not profess to treat diseases by, nor intend to practise, any exclusive system, generally recognized as such by the profession or declared so by the Councillors of the Society; and that he possesses such other qualifications as the Society may deem necessary.

II. Omit By-Law IX. in toto.

III. Change By-Law X. so as to read:

Any Fellow of this Society who advertises for sale, or otherwise offers to the public, any medicine the composition of which he keeps secret, or who offers to treat any disease by any such secret medicine, or by any secret treatment, shall be liable to expulsion, or to such other penalty as a Board of Trial may have power to inflict.

Dr. Cheever moved to amend the above by inserting after the word "practice" the words "nor to assume the name of" so that the clause would read, "that he does not profess to treat diseases by, nor intend to practise, nor to assume the name of any exclusive system," etc.

After an animated discussion participated in by Drs. F. C. Shattuck, Cheever, Folsom, Harvey, Wheatley, Lambert, Thompson and F. B. Harrington, the amendment proposed by Dr. Cheever was not adopted, and the amendments as offered to the Councillors by its Committee were passed.

Dr. Harvey offered the following, which was adopted:

That the Society recommends the appointment of a Committee by the Councillors at their next meeting in October, with instructions to re-draft the existing By-Laws, to suggest such changes therein as the interests of the Society seem to require, and to report thereon at the meeting of the Council in February, 1907.

Papers were presented as follows:

DISEASES OF THE BILE PASSAGES, INCLUDING THE LIVER, GALL BLADDER AND PANCREAS.

I.—By Maurice H. Richardson, M.D., of Boston (read by Dr. D. F. Jones).

II.—By Elbridge G. Cutler, M.D., of Boston.

The papers were discussed by Drs. R. H. Fitz, S. J. Mixter, F. B. Harrington, F. B. Lund, of Boston, and C. E. Edson of Denver, Colo.

Dr. Walter A. Smith, of Springfield, read an account of the Removal of an Enormous Ovarian Tumor.

At 12 M. the Annual Discourse was delivered by Dr. John L. Hildreth, of Cambridge.

Voted, That the thanks of the Society be presented to the Orator for his very interesting address.

The following delegates from other State Medical Societies were present:

New Hampshire.—Dr. J. D. Holt.
Rhode Island.—Dr. W. A. Risk.
Connecticut.—Dr. A. B. Coleburn.

At 1 P.M. the Annual Dinner was served to eleven hundred Fellows.

FRANCIS W. GOSS,
Recording Secretary.

TREASURER'S REPORT.

Receipts.

April 15, 1905—Balance in the Treasury			$11,199 05
Assessments at the Annual Meeting		$995 00	
" paid to Treasurer		655 00	
" paid to District Treasurers:			
Barnstable	$135 00		
Berkshire	330 00		
Bristol North	235 00		
Bristol South	470 00		
Essex North	485 00		
Essex South	705 00		
Franklin	165 00		
Hampden	845 00		
Hampshire	300 00		
Middlesex East	280 00		
Middlesex North	535 00		
Middlesex South	1,610 00		
Norfolk	1,570 00		
Norfolk South	270 00		
Plymouth	370 00		
Suffolk	2,780 00		
Worcester	1,090 00		
Worcester North	315 00		
Total	$12,490 00	12,490 00	
Total Assessments		$14,140 00	14,140 00
Sale of tickets at Annual Dinner			14 00

Income.

Interest New England Trust Co.	115 58	
" Bay State Trust Co.	129 20	
" Franklin Savings Bank	37 93	
" Roxbury Institution for Savings	35 30	
" Provident Institution for Savings	35 30	
" Suffolk Savings Bank	35 30	
" Massachusetts 3½% Bonds	175 00	
" United States Bond	400 00	
" Annuties Mass. Hospital Life Ins. Co.	816 80	
Total	$1,780 41	1,780 41
Sale of electricity at Annual Meeting		45 00
Renewal of certificate of membership		50
Total Receipts		$27,178 96

Expenses.

Secretary's Expense:		
Postage, printing and stationery	$278 37	
Engrossing and rolls	23 00	
Stenographer	9 88	
Clerk	18 00	
Total	329 25	339 25
Librarian's Expense:		
Postage and other delivery of Communications	191 24	
Wrappers	14 00	
Total	205 24	205 24
Censors' Expense		666 00
Supervisors' Expense		18 92
Treasurer's Expense:		
Postage, stationery and printing	99 39	
Cards for catalogue	10 50	
Rent of safe, Bay State Trust Co.	10 00	
Clerks at Annual Meeting	30 00	
Carriage of accounts, Annual Meeting	2 50	
	152 39	152 39
District Treasurer's Expense		761 99
Exchange		80
Salaries		1,400 00
Rent		750 00
Annual Dividend		4,500 00
Annual Meeting:		
Printing, postage and envelopes	$84 62	
Clerk to Committee	20 00	
Rent of Mechanic Hall	600 00	
Police	8 00	
Boston Towel Supply Co	2 00	
Attendance in coat room	6 00	
De Silva Sign Co.	50 39	
New England Decorating Co.	15 00	
Music	40 00	
Florist	50 00	
Caterer	2,541 56	
Cigars	168 50	
Steriopticon	10 00	
Darkening windows	5 90	
Electricity and attendants	146 60	
Gas piping	44 16	
Plumbing	38 15	
Tables	40 00	
Blankets and pillows	20 00	
Medical and surgical supplies	17 09	
Amounts carried forward,	$3,907 97	$8,694 59

Amounts brought forward,	$3,907 97	$8,694 59	
Photographic supplies	10 00		
Fees to patients	10 00		
Carriages	13 00		
Express	10 75		
Incidentals, Pathological Exhibit	3 05		
Incidentals, Bacteriological Exhibit	9 23		
Other incidentals	128 25		
Total	4,092 25	4,092 25	
Shattuck Lecture		200 00	
Cotting Lunch		106 80	
Catalogue:			
Clerical work	100 00		
Printing and mailing	632 78		
Bookbinding	25 40		
	758 18	758 18	
Committee To Procure Scientific Papers		60 50	
Committee On Publications—printing		1,007 41	
Committee On Diplomas		12 00	
Committee On State and National Legislation		120 20	
Committee On Ethics		21 50	
Committee On By-Laws		8 50	
Board of Trial:			
Fees and mileage	19 44		
Witness fees	30 00		
Legal fees	200 00		
	249 44	249 44	
Annual Meeting of 1906:			
Clerk	15 00		
Printed postal cards	70 00		
Envelopes	38 60		
	123 00	123 00	
Total Expenses		$15,544 37	15,544 37
Balance			$11,634 59

This balance is deposited as follows:

Deposit, New England Trust Co.	$5,490 88	
Less outstanding checks	356 00	
	5,134 88	
Deposit, Bay State Trust Co.	6,464 41	
Undrawn interest, Suffolk Savings Bank	35 30	
	$11,634 59	

This undrawn interest became available only on the day that the books were closed, and was shortly after withdrawn and deposited with the New England Trust Co.

Permanent Fund.

The permanent fund is the same as a year ago, and consists as follows :

Deposit Roxbury Institution for Savings, Cotting Fund,	$1,000 00
" Provident Institution for Savings, Cotting Fund	1,000 00
" Suffolk Savings Bank, Cotting Fund . . .	1,000 00
U. S. four per cent. bond, Phillips Fund . . .	10,000 00
Annuity bond, Mass. Hospital Life Ins. Co., Shattuck Fund,	9,166 87
" " Mass. Hospital Life Ins. Co. . . .	11,253 30
Massachusetts three and a half per cent. bonds . .	5,000 00
Deposit Franklin Savings Bank	1,074 48
Total . .	$39,494 65

EDWARD M. BUCKINGHAM,
Treasurer.

April 15, 1906.

The undersigned, a duly appointed committee, having examined the books of the treasurer, find them correctly cast and properly vouched, and also that he has in his possession the securities called for.

M. P. SMITHWICK,
Chairman.

May, 1906.

Officers of The Massachusetts Medical Society.

1906—1907.

CHOSEN JUNE 12, 1906.

GEORGE W. GAY, ...	Newton, .	PRESIDENT.
LEONARD WHEELER, . . .	Worcester, .	VICE-PRESIDENT.
FRANCIS W. GOSS, ...	Roxbury, .	COR. SECRETARY. REC. SECRETARY.
EDWARD M. BUCKINGHAM, .	Boston, . .	TREASURER.
EDWIN H. BRIGHAM, . . .	Brookline, .	LIBRARIAN.

Standing Committees.

Of Arrangements.

A. P. PERRY, L. W. GILBERT,
G. S. C. BADGER, C. C. SIMMONS,
LINCOLN DAVIS, G. H. FRANCIS.

On Publications.

O. F. WADSWORTH, G. B. SHATTUCK, H. L. BURRELL.

On Membership and Finance.

F. W. GOSS, WALTER ELA, C. M. GREEN,
A. COOLIDGE, JR., H. A. WOOD.

To Procure Scientific Papers.

J. B. BLAKE, B. P. CROFT, R. C. LARRABEE,
M. D. CLARKE, CHARLES HARRINGTON.

On Ethics and Discipline.

C. G. CARLETON, EDWARD COWLES, J. F. A. ADAMS,
LEONARD WHEELER, J. A. GAGE.

On Medical Diplomas.

H. E. MARION, O. F. ROGERS, H. W. NEWHALL.

On State and National Legislation.

G. W. GAY, H. P. BOWDITCH, S. D. PRESBREY,
G. G. SEARS, D. D. GILBERT.

Presidents of District Societies—Vice-Presidents (Ex-Officiis.)

[Arranged according to seniority.]

Abner Post,	J. H. Gifford,
T. M. Durell,	E. H. Guild,
J. F. Welch,	H. G. Blake,
C. C. Messer,	W. J. Sullivan,
David Harrower,	T. J. Robinson,
A. P. Mason,	H. B. Perry,
W. J. Sleeper,	A. B. Withington,
F. G. Wheatley,	H. W. Mitchell,
Samuel Crowell,	C. E. Harris.

Councillors.

Barnstable.—R. H. Faunce, Sandwich; W. D. Kinney, Osterville.

Berkshire.—J. F. A. Adams, Pittsfield; O. J. Brown, H. Bushnell, North Adams; H. Colt, I. S. F. Dodd, Pittsfield; A. T. Wakefield, Sheffield.

Bristol North.—A. S. Deane, W. Y. Fox, S. D. Presbrey, Taunton; A. M. Round, Norton.

Bristol South.—A. W. Buck, W. A. Dolan, S. V. Merritt, H. G. Wilbur, Fall River; H. C. Allen, C. F. Connor, S. W. Hayes, G. DeN. Hough, New Bedford.

Essex North.—G. M. Atwood, Bradford; J. F. Burnham, Lawrence; I. J. Clarke, Haverhill; J. A. Douglass, Amesbury; J. A. Hogan, Lawrence; E. H. Noyes, W. W. Pillsbury, Newburyport; R. B. Root, Georgetown; L. J. Young, Haverhill.

Essex South. G. G. Bailey, Ipswich; C. H. Bangs, Lynn; S. P. F. Cook, Gloucester; T. Dwight, Nahant; W. W. Eaton, Danvers: G. H. Gray, Lynn; H. J. Hall, Marblehead; T. Kittredge, Salem; W. B. Little, Lynn; H. E. Sears, J. A. Shatswell, Beverly; J. Shanahan, Peabody; F. E. Stone, Lynn; B. R. Symonds, Salem; A. M. Tupper, Rockport.

FRANKLIN.—H. G. Stetson, G. B. Twitchell, Greenfield; N. P. Wood, Northfield.

HAMPDEN.—C. A. Allen, Holyoke; R. P. M. Ames, T. S. Bacon, E. A. Bates, F. W. Chapin, Springfield; L. J. Gibbs, Chicopee Falls; C. W. Jackson, Monson; G. H. Janes, Westfield; D. E. Keefe, C. F. Lynch, H. C. Martin, W. A. Smith, F. B. Sweet, Springfield; J. P. Schneider, Palmer.

HAMPSHIRE.—A. G. Blodgett, Ware; F. C. Bruce, Easthampton; H. T. Shores, Northampton; W. P. Sutson, Cummington; E. D. Williams, Easthampton.

MIDDLESEX EAST.—A. H. Cowdrey, Stoneham; R. T. Edes, S. H. Parks, Reading; G. N. P. Mead, Winchester; F. E. Park, Stoneham.

MIDDLESEX NORTH.—C. M. Frye, A. E. Gillard, Lowell; A. Howard, Chelmsford; W. M. Jones, W. P. Lawler, J. B. O'Connor, C. E. Simpson, E. J. Welch, Lowell; J. W. Nichols, Tewksbury; F. E. Varney, North Chelmsford.

MIDDLESEX SOUTH.—T. Chamberlin, Concord; N. F. Chandler, Medford; J. M. Crocker, Cambridge; T. M. Durell, D. S. Flewelling, Somerville; G. W. Gay, *President*, Chestnut Hill; W. Ela, J. L. Hildreth, Cambridge; E. G. Hoitt, Marlboro'; A. A. Jackson, Everett; C. D. McCarthy, Malden; J. A. McDonald, Charlestown; H. E. Marion, Brighton; J. A. Mead, Watertown; G. A. Miles, Somerville; J. T. G. Nichols, W. Preble, Cambridge; C. E. Prior, G. Ryder, Malden; H. S. Rowen, Brighton; C. D. Sawin, Charlestown; F. M. Sherman, L. R. Stone, Newton; F. G. Smith, Somerville; I. E. Stowe, Medford; J. E. Somers, E. H. Stevens, F. W. Taylor, H. P. Walcott, Cambridge; W. H. Sylvester, Natick; J. Tolman, Arlington; A. M. Wilkins, South Framingham; H. A. Wood, A. Worcester, Waltham.

NORFOLK.—D. N. Blakely, Roxbury; B. H. Calkins, Jamaica Plain; H. L. Chase, F. W. Draper, G. H. Francis, Brookline; S. W. Crittenden, C. G. Dewey, D. D. Gilbert, J. S. Greene, Dorchester; D. Graham, R. W. Hastings, Brookline; H. R. Hitchcock, Hyde Park; C. D. Knowlton, Roxbury; W. C. Kite,

Milton; F. D. Lyon, A. A. Macdonald, E. G. Morse, Roxbury; H. T. Mansfield, Needham; M. L. Marr, Dorchester; M. V. Pierce, Milton; J. W. Pratt, Dedham; O. F. Rogers, Dorchester; W. H. Robinson, T. Smith, Jamaica Plain; H. R. Stedman, Brookline; H. B. Stevens, S. E. Stevens, West Roxbury; J. P. Treanor, Dorchester; E. Thayer, W. A. White, Roxbury; A. M. Worthington, Dedham.

NORFOLK SOUTH.—O. H. Howe, Cohasset; N. S. Hunting, F. E. Jones, Quincy; E. N. Mayberry, South Weymouth.

PLYMOUTH.—J. H. Averill, Campello; H. W. Dudley, Abington; N. K. Noyes, Duxbury; G. Osgood, Rockland; A. E. Paine, Brockton; F. G. Wheatley, North Abington.

SUFFOLK.—H. D. Arnold, J. B. Ayer, E. G. Brackett, E. H. Bradford, E. M. Buckingham, *Treasurer*, H. L. Burrell, J. F. Bush, A. T. Cabot, D. W. Cheever, A. Coolidge, Jr., F. J. Cotton, W. T. Councilman, E. G. Cutler, H. Derby, R. H. Fitz, C. F. Folsom, W. W. Gannett, C. M. Green, F. B. Harrington, H. Jackson, B. J. Jeffries, G. F. Jelly, E. P. Joslin, F. I. Knight, H. A. Lothrop, A. L. Mason, J. H. McCollom, A. B. Morong, J. G. Mumford, J. C. Munro, A. H. Nichols, C. B. Porter, A. Post, C. P. Putnam, J. J. Putnam, W. L. Richardson, T. M. Rotch, G. H. M. Rowe, J. D. K. Sabine, G. G. Sears, F. C. Shattuck, G. B. Shattuck, A. K. Stone, J. B. Swift, H. F. Vickery, O. F. Wadsworth, J. C. Warren, G. H. Washburn, J. C. White, H. Williams, G. Wolcott, Boston; E. S. Boland, M. F. Gavin, South Boston; J. M. Putnam, Chelsea.

WORCESTER.—W. P. Bowers, Clinton; P. P. Comey, J. T. Duggan, O. H. Everett, G. E. Francis, H. Gage, T. H. Gage, A. C. Getchell, R. W. Greene, D. Harrower, Worcester; E. B. Harvey, Westboro'; S. P. Holbrook, East Douglas; E. W. Norwood, Spencer; W. G. Reed, Southbridge; H. M. Quinby, E. V. Scribner, L. Wheeler, *Vice-President*, A. Wood, S. B. Woodward, Worcester.

WORCESTER NORTH.—C. E. Bigelow, Leominster; S. E. Greenwood, Templeton; H. H. Lyons, E. P. Miller, F. H. Thompson, Fitchburg; E. A. Sawyer, Gardner.

Censors.

BARNSTABLE.—S. T. Davis, Orleans; R. H. Faunce, *Supervisor*, Sandwich; S. F. Haskins, Cotuit; J. H. Higgins, Marston's Mills; E. M. Parker, South Yarmouth.

BERKSHIRE.—J. F. A. Adams, *Supervisor*, Pittsfield; M. M. Brown, H. Bushnell, North Adams; W. M. Mercer, Pittsfield; W. W. Scofield, Dalton.

BRISTOL NORTH.—H. B. Baker, Dighton; F. A. Hubbard, J. D. Mehegan, J. B. Murphy, S. D. Presbrey, *Supervisor*, Taunton.

BRISTOL SOUTH.—S. W. Bowen, A. I. Connell, W. A. Dolan, *Supervisor*, Fall River; C. A. Pratt, A. P. Webber, New Bedford.

ESSEX NORTH.—R. M. Birmingham, W. H. Merrill, Lawrence; J. A. FitzHugh, Amesbury; F. E. Sweetsir, Merrimac; L. J. Young, *Supervisor*, Haverhill.

ESSEX SOUTH.—F. W. Baldwin, Danvers; M. A. Harmon, Lynn; P. L. Sanborn, Marblehead; H. E. Sears, *Supervisor*, Beverly; S. C. Tucker, Peabody.

FRANKLIN.—E. G. Best, Turner's Falls; J. W. Cram, Colrain; G. R. Fessenden, Ashfield; C. G. Trow, Sunderland; N. P. Wood, *Supervisor*, Northfield.

HAMPDEN.—A. L. Damon, North Wilbraham; J. E. Marsh, D. E. Keefe, *Supervisor*, H. W. VanAllen, W. R. Weiser, Springfield.

HAMPSHIRE.—C. A. Byrne, Hatfield; A. M. Belden, J. C. Fahey, Northampton; D. M. Ryan, Ware; H. T. Shores, *Supervisor*, Northampton.

MIDDLESEX EAST.—M. A. Cummings, Winchester; G. F. Dow, Reading; E. S. Jack, Melrose; W. H. Keleher, Woburn; F. E. Park, *Supervisor*, Stoneham.

MIDDLESEX NORTH.—T. G. McGannon, J. V. Meigs, G. E. Pinkham, O. P. Porter, C. E. Simpson, *Supervisor*, Lowell.

MIDDLESEX SOUTH.—G. A. Bancroft, South Natick; H. D. Chadwick, Waltham; L. M. Palmer, South Framingham; W. Preble, *Supervisor*, Cambridge; H. S. Rowen, Brighton.

NORFOLK.—G. H. Francis, *Supervisor*, Brookline; E. T. Galligan, Roxbury; G. H. Ingalls, Jamaica Plain; R. M. Merrick, E. T. Twitchell, Dorchester.

NORFOLK SOUTH.—G. D. Bullock, Weymouth; H. L. Dearing, Braintree; R. McLellan, Quincy; E. N. Mayberry, *Supervisor*, V. M. Tirrell, South Weymouth.

PLYMOUTH.—J. E. Bacon, W. P. Chisholm, Brockton; N. C. King, Campello; A. A. MacKeen, Whitman; G. Osgood, *Supervisor*, Rockland.

SUFFOLK.—F. G. Balch, J. N. Coolidge, W. F. Gay, C. H. Hare, A. K. Stone, *Supervisor*, Boston.

WORCESTER.—F. H. Baker, W. T. Clark, M. F. Fallon, E. V. Scribner, *Supervisor*, Worcester; F. H. Clapp, North Grafton.

WORCESTER NORTH.—J. B. Donnelly, West Gardner; E. P. Miller, *Supervisor*, C. W. Spring, Fitchburg; A. E. Mossman, Westminster; A. H. Pierce, Leominster.

Commissioners of Trials.

BARNSTABLE	F. A. Binford	Hyannis.
BERKSHIRE	A. T. Wakefield	Sheffield.
BRISTOL NORTH	E. Washburn	Taunton.
BRISTOL SOUTH	G. L. Richards	Fall River.
ESSEX NORTH	J. F. Young	Newburyport.
ESSEX SOUTH	J. Allen	Topsfield.
FRANKLIN	J. D. Seymour	Whately.
HAMPDEN	J. W. Hannum	Ludlow.
HAMPSHIRE	C. Seymour	Northampton.

Middlesex East	D. T. Buzzell	Wilmington.
Middlesex North	G. A. Harlow	Tyngsboro'.
Middlesex South	J. F. Frisbie	Newton.
Norfolk	W. Noyes	Mattapan.
Norfolk South	G. W. Tinkham	Weymouth.
Plymouth	C. A. Drew	State Farm.
Suffolk	M. F. Gavin	Boston.
Worcester	C. H. Perry	Worcester.
Worcester North	L. G. Chandler	Townsend.

Officers of the District Medical Societies.

Barnstable.—C. E. Harris, Hyannis, *President;* C. M. Baker, Hyannis, *Vice-President;* H. B. Hart, East Dennis, *Secretary;* C. W. Milliken, Barnstable, *Treasurer.*

Berkshire.—A. B. Withington, Pittsfield, *President;* G. L. Rice, North Adams, *Vice-President;* I. S. F. Dodd, Pittsfield, *Secretary;* W. L. Paddock, Pittsfield, *Treasurer.*

Bristol North.—T. J. Robinson, Taunton, *President;* E. Washburn, Taunton, *Vice-President;* R. D. Dean, Taunton, *Secretary;* W. Y. Fox, Taunton, *Treasurer;* T. F. Clark, Taunton, *Librarian.*

Bristol South.—J. H. Gifford, Fall River, *President;* G. deN. Hough, New Bedford, *Vice-President;* A. J. Abbe, Fall River, *Secretary and Treasurer.*

Essex North.—W. J. Sullivan, Lawrence, *President;* C. E. Durant, Haverhill, *Vice-President;* M. D. Clarke, Haverhill, *Secretary and Treasurer.*

Essex South.—H. W. Mitchell, Hathorne, *President;* P. C. Proctor, Gloucester, *Vice-President;* G. K. Blair, Salem, *Secretary;* G. Z. Goodell, Salem, *Treasurer;* A. M. Patterson, Peabody, *Librarian.*

Franklin.—C. C. Messer, Turner's Falls, *President;* B. P. Croft, Greenfield, *Vice-President;* C. F. Canedy, Greenfield, *Secretary and Treasurer.*

HAMPDEN.—E. H. Guild, Springfield, *President;* D. F. Donoghue, Holyoke, *Vice-President;* F. S. Ward, Springfield, *Secretary and Treasurer.*

HAMPSHIRE.—H. B. Perry, Northampton, *President;* J. G. Hayes, Williamsburg, *Vice-President;* A. G. Minshall, Northampton, *Secretary;* A. H. Hoadley, Northampton, *Treasurer;* E. W. Brown, Northampton, *Librarian.*

MIDDLESEX EAST.—H. G. Blake, Woburn, *President;* C. W. Harlow, Melrose Highlands, *Vice-President;* H. A. Gale, Winchester, *Secretary;* C. Dutton, Wakefield, *Treasurer.*

MIDDLESEX NORTH.—W. J. Sleeper, Westford, *President;* W. A. Johnson, Lowell, *Vice-President;* R. J. Meigs, Lowell, *Secretary;* T. B. Smith, Lowell, *Treasurer;* D. N. Patterson, Lowell, *Librarian.*

MIDDLESEX SOUTH.—T. M. Durrell, Somerville, *President;* C. E. Prior, Malden, *Vice-President;* F. R. Jouett, Cambridge, *Secretary;* W. Ela, Cambridge, *Treasurer;* E. S. Dow, Allston, *Librarian.*

NORFOLK.—S. Crowell, Dorchester, *President;* A. P. Perry, Jamaica Plain, *Vice-President;* J. C. D. Pigeon, Roxbury, *Secretary and Librarian;* E. G. Morse, Roxbury, *Treasurer.*

NORFOLK SOUTH.—J. F. Welch, Quincy, *President;* N. S. Hunting, Quincy, *Vice-President;* C. S. Adams, Wollaston, *Secretary, Treasurer and Librarian.*

PLYMOUTH.—F. G. Wheatley, North Abington, *President;* F. J. Ripley, Brockton, *Vice-President;* A. C. Smith, Brockton, *Secretary and Treasurer;* C. E. Lovell, Whitman, *Librarian.*

SUFFOLK.—Abner Post, Boston, *President;* C. P. Putnam, Boston, *Vice-President;* L. R. G. Crandon, Boston, *Secretary;* W. H. Prescott, Boston, *Treasurer;* B. J. Jeffries, Boston, *Librarian.*

WORCESTER.—D. Harrower, Worcester, *President;* E. V. Scribner, Worcester, *Vice-President;* G. E. Emery, Worcester, *Secretary;* G. O. Ward, Worcester, *Treasurer;* M. Lincoln, Worcester, *Librarian.*

WORCESTER NORTH.—A. P. Mason, Fitchburg, *President*: A. E. Mossman, Westminster, *Vice-President;* W. F. Sawyer, Fitchburg, *Secretary;* E. L. Fiske, Fitchburg, *Treasurer;* A. P. Mason, Fitchburg, *Librarian.*

The Massachusetts Medical Society.

PROCEEDINGS OF THE COUNCILLORS.

OCTOBER 3, 1906.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, October 3, 1906, at 12, noon.

The President, Dr. GEORGE W. GAY, in the chair.

The following Councillors were present:

Berkshire.
J. F. A. Adams,
I. S. F. Dodd.

Bristol North.
S. D. Presbrey.

Bristol South.
A. W. Buck,
W. A. Dolan,
G. deN. Hough,
H. G. Wilbur.

Essex North.
J. F. Burnham,
I. J. Clarke,
E. H. Noyes,
W. W. Pillsbury,
L. J. Young.

Essex South.
S. P. F. Cook,
T. Dwight,
H. E. Sears,
A. M. Tupper.

Hampden.
T. S. Bacon,
C. W. Jackson,
G. H. Janes,
J. P. Schneider.

Hampshire.
A. G. Blodgett.

Middlesex East.
R. T. Edes,
F. E. Park.

Middlesex North.
A. E. Gillard,
J. B. O'Connor.

Middlesex South.
T. M. Durell,
W. Ela,
G. W. Gay,
J. L. Hildreth,
A. A. Jackson,
H. E. Marion,
J. T. G. Nichols,
W. Preble,
C. E. Prior,
G. Ryder,
F. M. Sherman,
F. G. Smith,
J. E. Somers,
E H. Stevens,
L. R. Stone,
I.. E. Stowe,
W. H. Sylvester,
F. W. Taylor,
J. Tolman,
H. P. Walcott,
A. M. Wilkins.

Norfolk.
H. L. Chase,
S. W. Crittenden,
G. H. Francis.
D. D. Gilbert,
D. Graham,
R. W. Hastings,
W. C. Kite,
H. T. Mansfield,
E. G. Morse,

M. V. Pierce,
J. W. Pratt,
W. H. Robinson,
T. Smith,
E. Thayer.

Norfolk South.

E. N. Mayberry.

Suffolk.

H. D. Arnold,
E. S. Boland,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
F. J. Cotton,
E. G. Cutler,
R. H. Fitz,
C. F. Folsom,
C. M. Green,
F. B. Harrington,
E. P. Joslin,
C. B. Porter,
C. P. Putnam,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
J. D. K. Sabine,
G. G. Sears,
F. C. Shattuck,
G. B. Shattuck,
A. K. Stone,
J. B. Swift,
H. F. Vickery,
H. Williams,
G. Wolcott.

Worcester.

G. E. Francis,
A. C. Getchell.
R. W. Greene,
E. B. Harvey,
E. W. Norwood.

Worcester North.

E. A. Sawyer,
F. H. Thompson.

Total, 94.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other State Medical Societies:

Vermont.—H. D. Arnold, of Boston; I. S. F. Dodd, of Pittsfield.

New York.—J. L. Hildreth, of Cambridge; W. H. Pomeroy, of Springfield.

The Committee on Membership and Finance reported, and in accordance with their recommendation it was voted that the resignations of the following be accepted:

Donald Churchill, of Providence, R. I.
Charles W. Eveleth, of New York, N. Y.
Sherwin Gibbons of Los Angeles, Cal.
Edward S. Hatch, of New Orleans, La.
Beverly MacMonagle, of San Francisco, Cal.
George W. Nash, of Hurley, N. Y.
Russell S. Rowland of Detroit, Mich.
Alice W. Tallant, of Philadelphia, Pa.

Also that the following be placed on the retired list:

John P. Brown, of Taunton.
Edward R. Cogswell, of Cambridge.
Octavius B. Shreve, of Salem.

Also that the following be deprived of the privileges of membership for non-payment of dues:

Frederick A. Davis, of Boston.

Charles F. Denny, of St. Paul, Minn.
John A. Elmere, of Los Angeles, Cal.
Emma L. Erving, of Washington, D. C.
William G. Erving, of Washington, D. C.
Ammen Farenholt, U. S. Navy.
Nathan L. Griffin, of Bradford, N. H.
Harold J. Levis, of Rochester, N. Y.
Dwight M. Lewis, of New Haven, Conn.
George F. Lewis, of New Bedford.
Philippe H. Nichol, of Worcester.
Clarence C. Northrop, of Manchester, N. H.
Payn B. Parsons, of New York, N. Y.
Archibald J. Ranney, of Cleveland, O.
Hiram H. Seelye, of Daytona, Fla.
Kenelm Winslow, of Seattle, Wash.

It was also recommended by the Committee, and it was voted, That for the current year, and until otherwise ordered, the premium on the Treasurer's bond be paid by the Society.

Voted, That the following be restored to the privileges of membership:

Bradford Allen, of Nashua, N. H.
Winfred Carle Pike, of Boston.

The Secretary reported the following vote from the last annual meeting of the Society:

That the Society recommends the appointment of a Committee by the Councillors at their next meeting in October, with instructions to re-draft the existing By-Laws, to suggest such changes therein as the interests of the Society seem to require, and to report therein at the meeting of the Council in February, 1907.

Voted, That a Committee of five be appointed to revise the By-Laws in accordance with the recommendations of the Society.

The following were appointed to constitute the Committee: E. B. Harvey, F. C. Shattuck, F. W. Goss, G. deN. Hough, E. L. Peirson.

Dr. Cabot spoke of the desire on the part of the American Medical Association that a Committee On Medical Education be established by each State Medical Society, and in furtherance thereof offered the following, which was adopted:

Voted, To establish a Committee On Medical Education to consist of three members.

This Committee is to be appointed in the same manner as the other standing Committees.

It shall be the duty of this Committee to promote the cause of higher medical education in all possible ways. It may lend its assistance to the State Examining Board and may co-operate with the Council on Medical Education of the American Medical Association for the advancement of this object.

It shall report to the Council each year at the October meeting.

Voted, That the following constitute the Committee:

Drs. E. H. Bradford, H. C. Ernst, and H. D. Arnold.

Adjourned at 12.35 P.M.

FRANCIS W. GOSS,
Recording Secretary.

FEBRUARY 6, 1907.

A STATED MEETING of the Councillors was held in the Medical Library, Boston, on Wednesday, February 6, 1907, at 12, noon.

The President, Dr. GEORGE W. GAY, in the chair.

The following Councillors were present:

Berkshire.
J. F. A. Adams.

Bristol South.
G. deN. Hough.

Essex North.
J. F. Burnham,
E. H. Noyes,
W. W. Pillsbury,
L. J. Young.

Essex South.
C. H. Bangs,
G. H. Gray,
B. R. Symonds.

Franklin.
G. P. Twitchell.

Hampden.
R. P. M. Ames,
T. S. Bacon,
G. H. Janes.

Hampshire.
F. C. Bruce,
E. D. Williams.

Middlesex East.
R. T. Edes,
G. N. P. Mead,
S. H. Parks.

Middlesex North.
J. H. Nichols.

Middlesex South.
J. M. Crocker,
G. W. Gay,
A. A. Jackson,
H. E. Marion,
E. H. Stevens,
L. R. Stone,
I. E. Stowe,
W. H. Sylvester,
F. W. Taylor,
J. Tolman.

Norfolk.
D. N. Blakely,
B. H. Calkins,
F. W. Draper,
G. H. Francis,
D. D. Gilbert,
D. Graham,
J. S. Greene,
H. R. Hitchcock,
E. G. Morse,
W. H. Robinson,
T. Smith,
H. B. Stevens,
S. E. Stevens,
E. Thayer,
J. P. Treanor,
A. M. Worthington.

Norfolk South.
F. E. Jones.

Plymouth.
A. E. Paine,
F. G. Wheatley.

Suffolk.
H. D. Arnold,
J. B. Ayer,
E. S. Boland,
E. G. Brackett,
E. M. Buckingham,
J. F. Bush,
A. T. Cabot,
D. W. Cheever,
A. Coolidge, Jr.,
R. H. Fitz,
C. F. Folsom,
C. M. Green,
F. B. Harrington,
E. P. Joslin,
A. L. Mason,
A. B. Morong,
J. G. Mumford,
C. B. Porter,
C. P. Putnam,
J. M. Putnam,
W. L. Richardson,
G. H. M. Rowe,
G. G. Sears,
F. C. Shattuck,
G. B. Shattuck,
A. K. Stone,
J. C. Warren,
J. C. White,
H. Williams,
G. Wolcott.

Worcester.
O. H. Everett,
H. Gage,
R. W. Greene,
D. Harrower,
E. B. Harvey,
S. P. Holbrook,
S. B. Woodward.

Worcester North.
F. H. Thompson.

Total, 86.

The record of the last meeting was read and accepted.

The following were appointed Delegates to other Medical Societies:

American Medical Association, 1907 and 1908.—C. H. Cook, of Natick; D. J. Brown, of Springfield; R. C. Cabot, of Boston.

Alternates.—P. P. Comey, of Worcester; Henry Colt, of Pittsfield; C. A. Porter, of Boston.

Maine.—Harold Williams, of Boston; B. R. Symonds, of Salem.

New Hampshire.—W. E. Faulkner, of Boston; J. V. Meigs, of Lowell.

Rhode Island.—S. B. Woodward, of Worcester; F. J. Cotton, of Boston.

Connecticut.—G. W. W. Brewster, of Boston; L. H. Plimpton, of Norwood.

New Jersey.—J. H. McCollom, J. B. Blake, of Boston.

The following Committee was appointed:

To Audit the Treasurer's Accounts.—E. A. Locke, M. V. Pierce.

The Committee on Membership and Finance reported, and in accordance with their recommendation it was voted that the resignations of the following be accepted:

James T. Fisher, of Los Angeles, Cal.
James F. Hall, U. S. Army.

Also that the following be placed on the retired list:

David W. Cheever, of Boston.
Samuel A. Davis, of Somerville.
Eugene A. Gilman, of Roxbury.
Horace P. Makechnie, of West Somerville.

Also that the following be deprived of the privileges of membership for non-payment of dues:

Charles E. Bedell, of Somerville.
Joseph T. L. Brennan, of Lowell.
Daniel R. Brown, of Salem.
Thomas F. Carroll, of Boston.
William E. Carroll, of Taunton.

Robert R. Culbert, of Roxbury.
Charles H. Curran, of Holyoke.
Newbert J. Hall, of Brookline.
Alvin D. Holmes, of Hyde Park.
George P. Hurd, of Holbrook.
Thomas L. Jenkins, of Topsfield.
James R. Matheson, of Roxbury.
Laura T. Myers, of Allston.
Wilbur B. Robertson, of Worcester.
Frank R. Searles (residence unknown).
George A. Spencer, of Haverhill.
Maurice J. Treston (residence unknown).

The Committee also reported recommending the election to honorary membership of Beverly MacMonagle, M.D., of San Francisco, Cal., whose nomination to that position was referred to them at the last meeting.

By ballot, Beverly MacMonagle, M.D., was elected an honorary member of the Society.

Dr. Harvey, for the Committee appointed at the last meeting to re-draft the existing By-Laws, reported a proposed code for the consideration of the Councillors, and to receive suggestions from them with the view of a subsequent report at the annual meetings of the Council and Society.

The code was considered by sections, *seriatim*, and numerous suggestions were made. On the question of changing the ratio of Councillors to the membership of the District Societies, it was suggested that a circular letter be sent to these Societies, requesting an expression of opinion on this matter.

The Councillors also adopted a series of "Special Rules of the Council," as presented by the Committee.

Adjourned at 2 P.M.

FRANCIS W. GOSS,
Recording Secretary.

JUNE 11, 1907.

THE ANNUAL MEETING of the Councillors was held in Mechanic Building, Boston, on Tuesday, June 11, 1907, at 5 P.M.

The President, Dr. GEORGE W. GAY, in the chair.

The following Councillors were present:

Barnstable.
H. B. Hart.

Berkshire.
O. J. Brown,
I. S. F. Dodd.

Bristol North.
A. S. Deane,
F. A. Hubbard.

Bristol South.
J. A. Barré,
A. W. Buck,
W. A. Dolan,
H. G. Wilbur.

Essex North.
J. F. Burnham,
I. J. Clarke,
J. A. Douglass,
W. W. Pillsbury,
J. W. Rand,
L. J. Young.

Essex South.
C. H. Bangs,
S. P. F. Cooke,
P. P. Johnson,
H. W. Mitchell,
G. E. MacArthur,
P. C. Proctor,
H. E. Sears,
J. A. Shatswell.

Franklin.
H. G. Stetson,
G. P. Twitchell.

Hampden.
C. W. Jackson,
H. G. Webber.

Hampshire.
A. M. Belden,
O. W. Cobb,
N. Holden.

Middlesex North.
W. P. Lawler,
C. E. Simpson,
W. J. Sleeper.

Middlesex South.
T. M. Durell,
W. Ela,
D. S. Flewelling,
G. W. Gay,
E G. Hoitt,
H. E. Marion,
J. T. G. Nichols,
L. M. Palmer,
C. E. Prior,
G. Ryder,
F. M. Sherman,
E. H. Stevens,
L. R. Stone,
I. E. Stowe,
W. H. Sylvester,
F. W. Taylor,
A. M. Wilkins.

Norfolk.
B. S. Blanchard,
S. W. Crittenden,
M. J. Cronin,
C. B. Darling,
C. G. Dewey,
D. D. Gilbert,
J. S. Greene,
T. F. Greene,
H. R. Hitchcock,
H. T. Holland,
G. N. Jones,
H. T. Mansfield,
E. G. Morse,
M. V. Pierce,
J. W. Pratt,
W. H. Robinson,
T. Smith,
W. A. White.
C. H. Winn.

Norfolk South.
T. J. Dion,
R. McLellan,
W. H. W. Sturgis,
V. M. Tirrell.

Plymouth.
J. H. Averill,
G. Osgood,
A. E. Paine.

Suffolk.
H. D. Arnold,
J. B. Ayer,
E. S. Boland,
E. H. Bradford,
E. M. Buckingham,
H. L. Burrell,
J. F. Bush,
A. T. Cabot,
A. Coolidge, Jr.,
R. H. Fitz,

C. M. Green,
F. B. Harrington,
B. J. Jeffries,
J. H. McCollom,
A. B. Morong,
J. G. Mumford,
J. C. Munro,
A. H. Nichols,
C. B. Porter,
A. Post,
J. M. Putnam,
W. L. Richardson,
T. M. Rotch,
J. D. K. Sabine,
G. G. Sears,
F. C. Shattuck,
G. B. Shattuck,
A. K. Stone,
H. F. Vickery,
H. Williams,
G. Wolcott.

Worcester.

W. P. Bowers,
P. P. Comey,
O. H. Everett,
G. E. Francis,
A. C. Getchell,
D. Harrower,
E. B. Harvey,
S. P. Holbrook,
E. W. Norwood,
L. Wheeler,
L. F. Woodward,
S. B. Woodward.

Worcester North.

C. E. Bigelow,
E. P. Miller,
F. H. Thompson.

Total, 122.

The record of the last meeting was read and accepted.

The Secretary reported that 171 Fellows had been admitted since the last annual meeting, and that 37 deaths had been recorded.

The Treasurer, Dr. BUCKINGHAM, read his annual report.

The Auditing Committee reported that they found the accounts correctly cast and properly vouched, and that the Treasurer has in his possession the securities called for.

Voted, To accept the Treasurer's report.

The Committee on Membership and Finance reported, and recommended that $4,500.00 of the surplus in the treasury be distributed among the District Societies.

Voted, That the recommendation be adopted.

In accordance with the recommendation of the same Committee it was voted, that the resignations of the following be accepted:

Melvin P. Burnham, of Ray Brook, N. Y.
Francis J. Dore, of Roxbury.
George H. Gleason, of Dorchester.
Benjamin J. Handy, of Fall River.
Robert B. Harrington, of Northampton.
William E. MacCoy, of Brookline.
William R. May, of New York, N. Y.
Harry C. Parker, of Indianapolis, Ind.
Otis W. Sedgewick, of Brookfield, Conn.
Elmer S. Tenney, U. S. Army.

Also that the following be placed on the retired list:

Robert Amory, of Boston.
Charles B. Bridgham, of Cohasset.
John G. Dearborn, of Charlestown.
Robert Disbrow, of Boston.
William W. Eaton, of Danvers.
Hugh Ferguson, of South Boston.
Linnæus V. Gibbs, of Huntington.
Thomas Hall, of Boston.
Horace G. Leslie, of Amesbury.
William M. Mercer, of Pittsfield.
Albert L. Norris, of Cambridge.
Hamilton Osgood, of Boston.
Charles B. Porter, of Boston.
George E. Stackpole, of Malden.
Lincoln R. Stone, of Newton.

The Committee on Publications reported through Dr. Shattuck.

The Committee on State and National Legislation reported, through Dr. Sears:

That during the past year they have appeared either in person or by representative before the Committee of Public Health, advocating the passage of the following bills: For Labeling Patent and Proprietary Drugs and Foods; To Prohibit the Evasion of Vaccination Laws; To Regulate the Sale of Poisons; For Appointment of Medical Inspectors through the State; For Proper Sanitation of Factories; For the Transfer of Certain Powers from the District Police to the State Board of Health; and for further Provision for the Care of Patients with Advanced Tuberculosis. They have opposed the bill to create a State Board of Osteopathic Examination and Registration. The Auxiliary Legislative Committee has proved of great service in reaching certain members of the Legislature.

Voted, That the following be restored to the privileges of membership:

Francis Xavier Crawford, of Boston.
Edwin Putnam Gleason, of Brockton.

The Committee on Nominations reported a list of candidates for the offices of the Society for the ensuing year, and the same were elected by ballot:

President	GEORGE W. GAY, of Newton.
Vice-President	FRANCIS J. CANEDY, of Shelburne Falls.
Secretary	FRANCIS W. GOSS, of Roxbury.
Treasurer	EDWARD M. BUCKINGHAM, of Boston.
Librarian	EDWIN H. BRIGHAM, of Brookline.

THOMAS F. HARRINGTON, of Boston, was chosen Orator for the annual meeting of the Society in 1908.

Voted, That the next annual meeting of the Society be held in Boston on the second Wednesday in June, 1908.

The following Standing Committees were appointed:

Of Arrangements.

G. S. C. Badger, C. C. Simmons,
Lincoln Davis, G. H. Francis,
L. W. Gilbert, A. N. Broughton.

On Publications.

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

On Membership and Finance.

F. W. Goss, Walter Ela, C. M. Green,
A. Coolidge, Jr. H. A. Wood.

To Procure Scientific Papers.

B. P. Croft, R. C. Larrabee, M. D. Clarke,
Charles Harrington, R. B. Greenough.

On Ethics and Discipline.

C. G. Carleton, J. F. A. Adams, Leonard Wheeler,
J. A. Gage, J. W. Bartol.

On Medical Diplomas.

H. E. Marion, O. F. Rogers, H. W. Newhall.

On Medical Education.

H. C. Ernst, H. D. Arnold, C. H. Williams.

On State and National Legislation.

G. W. Gay, S. D. Presbrey, G. G. Sears,
D. D. Gilbert, C. H. Cook.

Voted, That the following be appointed to fill vacancies in Board of Censors:

For Essex South District: F. S. Atwood, H. W. Newhall.

For Worcester North District: H. W. Page, E. J. Tully.

Also to fill a vacancy in the Councillors for Middlesex North: J. B. O'Connor.

On motion of Dr. Cabot it was voted, That the sum of $300.00 be appropriated from the Society's treasury for the use of the Committee on Tuberculosis.

Voted, That $300.00 be appropriated for a memorial to Dr. N. S. Davis as the founder of the American Medical Association.

Dr. Gilbert moved, and it was voted, that the matter of Medical Expert Testimony reform, referred to the Council by the Norfolk District Medical Society, be referred to the Committee on State and National Legislation, to take such action as they consider best.

The President called attention to the request of the Committee on Medical Legislation of the American Medical Association, that a delegate be appointed from this Society in the National Legislative Council.

Voted, That Dr. S. D. Presbrey be appointed as the delegate.

Dr. Harvey, Chairman of the Committee on Revision of the By-Laws, presented a code revised to meet suggestions made at the last meeting of the Council, and requested further suggestions, if any changes were desirable. A slight amendment was made to By-Law 12, and it was voted that it was the desire of the Council that it be adopted.

Dr. Harvey desired an opinion as to the ratio of Councillors to membership in the District Societies.

The Secretary stated, as a result of the request that the District Societies express their desire in the matter, that of the 18 District Societies 10 had replied. Of these one wished the ratio to be 1 to 15; one, 1 to 16; and eight, 1 to 20.

It was then voted that 1 to 20, as embodied in the report, be recommended.

Dr. Harvey moved, and it was voted, that the Council commends the new draft of By-Laws submitted by the Committee on By-Laws, and orders that the same be reported by the Committee to the next annual meeting of the Society, for adoption.

Adjourned at 6.10 P.M.

FRANCIS W. GOSS,
Recording Secretary.

The Massachusetts Medical Society.

PROCEEDINGS OF THE SOCIETY.

ANNUAL MEETING.

First Day.

June 11, 1907.

Sectional meetings were held in Mechanic Building, Boston, on Tuesday, June 11, 1907, at 2 P.M.

The Sections were organized and papers were presented as follows:

SECTION IN SURGERY.

Dr. H. L. Burrell, Boston . . . . . Chairman.
Dr. J. Bapst Blake, Boston . . . . . Secretary.

Operative Treatment of Varicose Veins.—By Dr. J. T. Bottomley, of Boston.

The Prostate from the Surgical Standpoint.—By Dr. C. S. Benson, of Haverhill.

Lantern-slide demonstration of the facts concerning Stenosis of the Pylorus in Infancy; its Diagnosis and Treatment, with the Report of three Cases successfully operated upon.—By Dr. C. L. Scudder, of Boston.

Twenty-two years' experience in the Treatment of Stricture of the Urethra.—By Dr. F. S. Watson, of Boston.

SECTION IN MEDICINE.

Dr. G. G. Sears, Boston . . . . . Chairman.
Dr. R. C. Larrabee, Boston . . . . . Secretary.

Subacute Polymyositis, with a Report of three Cases.—By Dr. B. T. Burley, of Worcester.

OPSONINS AND BACTERIAL VACCINES.—By Drs. Timothy Leary and L. S. Medalia of Boston.

CLINICAL EXPERIENCE IN THE USE OF BACTERIAL VACCINES IN THE TREATMENT OF JOINT DISEASES, TUBERCULOUS AND NON-TUBERCULOUS.—By Dr. C. F. Painter, of Boston.

THE TREATMENT OF TYPHOID FEVER WITH SPECIFIC SERA, FILTRATES AND RESIDUES.—By Dr. M. W. Richardson, of Jamaica Plain.

SECTION ON TUBERCULOSIS.

Dr. A. T. CABOT, Boston . . . . . Chairman.
Dr. T. F. HARRINGTON, Boston . . . . . Secretary.

FACTORY INSPECTION.—By Dr. F. G. Wheatley, of North Abington.

HEALTH INSPECTORS: THEIR FUNCTION.—By Dr. Charles Harrington, of Boston.

THE CLASS METHOD FOR THE HOME TREATMENT OF CONSUMPTION, WITH DEMONSTRATION OF CASES, AND LANTERN-SLIDES.—By Dr. J. H. Pratt, of Boston.

THE CHANNELS OF INFECTION IN TUBERCULOSIS.—By Dr. Theobald Smith, of Boston.

During the sessions of the Society there was a Demonstration of Clinical and Laboratory Methods used in Medicine, organized by Dr. W. H. ROBEY, as follows:

1.	Pathology	Dr. F. B. Mallory.
2.	Rabies	Dr. Langdon Frothingham.
3.	Opsonic Index . . .	Dr. Timothy Leary.
4.	Blood	Dr. R. C. Larrabee.
5.	Urine	Dr. Adler.
6.	Stomach Contents . .	Dr. F. W. White.
7.	X-Ray	Dr. Percy Brown.
8.	Skin	Dr. H. P. Towle.
9.	Intestinal Parasites . .	Dr. Overlander.
10.	Board of Health Laboratory	Mr. Rickards.
11.	Boston Society for the Relief and Control of Tuberculosis .	Mr. A. M. Wilson.
12.	Medical Inspection of Schools.	

At 8 P.M. the Shattuck Lecture was delivered by FREDERICK C. SHATTUCK, M.D., of Boston.

Following the lecture a reception was given to the President by the Society.

SECOND DAY.

JUNE 12, 1907.

THE Society met in Mechanic Building, Boston, on Wednesday, June 12, 1907, at 9 A.M., for the exercises of the one hundred and twenty-sixth Anniversary.

The President, Dr. GEORGE W. GAY, in the chair.

The record of the last annual meeting was read and accepted.

The Secretary announced that 171 Fellows had been admitted during the past year and that 37 deaths had been recorded.

The following are the lists:

Admissions reported from June 12, 1906, to June 11, 1907.

1906	Adams, James Thacher	Boston.
1906	Bain, John Baxter	Lawrence.
1906	Bannon, John Hugh	Lawrence.
1907	Barrett, Edward William	Medford.
1906	Barron, Elmer Walter	Charlestown.
1906	Barstow, Andrew Thaddeus	Fitchburg.
1907	Bates, Lewis Beals	Boston.
1906	Beauchamp, Joseph Octave	Chicopee Falls.
1906	Beaulieu, Francis Xavier	Taunton.
1907	Beeley, Leon Gage	Lawrence.
1906	Bellehumeur, David Stanislaus	Lowell.
1907	Berwick, James Roderick	Methuen.
1907	Bessey, Earle Emerson	Hathorne.
1907	Bier, Max Daniel	Lawrence.
1907	Boothby, Walter Meredith	Boston.
1906	Boyle, Jeremiah Joseph	Cambridge.
1907	Brainerd, Walter Scott	Bradford.
1907	Bruce, Harold Milton	Brookline.
1907	Bullard, Edward Arthur	Cambridge.
1906	Burgess, Charles James	Lawrence.
1906	Bushold, Fred George	Lawrence.
1906	Campbell, Matthew Philip	Provincetown.
1906	Carley, Frederic James	New Bedford.
1906	Chalmers, Hattie Elizabeth	Boston.
1907	Champion, Merrill Edwin	Arlington.
1906	Chapin, Laurence Dudley	Springfield.
1906	Clancy, Daniel Francis	Hopkinton.

1907	Clark, William Irving, Jr.	Worcester.
1907	Cobb, Chester Thompson	Easthampton.
1906	Conlon, Frank Aloysius	Lawrence.
1907	Connor, William Henry	Springfield.
1906	Conroy, Edward Cornelius	Andover.
1907	Coon, William Hall	Lawrence.
1906	Corbett, Jeremiah Joseph	Boston.
1907	Coriat, Isidor Henry	Boston.
1906	Cornwall, Anna Marlin	Boston.
1907	Cotter, Edward Joseph	Boston.
1907	Cowan, Marion	Lynn.
1907	Cox, Ann Caroline	Jamaica Plain.
1907	Crispo, Peter Timothy	Fall River.
1906	Cusick, Lawrence Francis	Boston.
1906	Cyr, Emile Edouard	Lawrence.
1907	Dascombe, Otho Lee	Waltham.
1906	Davis, Ernest Leland	Springfield.
1906	Day, Cushman	Boston.
1907	Deacon, Thomas Irving	Cambridge.
1906	Deems, Owen Manfred	Springfield.
1906	Dewing, Louis Alexander	Boston.
1907	Dickinson, Gyrdie Willie	Jamaica Plain.
1907	Dowling, Charles Joseph	Springfield.
1906	Downing, Andrew Francis	Watertown.
1906	Dutra, Emmanuel Christian	Taunton.
1906	Eaton, William Edward	Roxbury.
1906	Felch, Carrie Innes	Boston.
1906	Felch, Lewis Perley	Boston.
1907	Finegan, Daniel Joseph	Gloucester.
1906	Frothingham, Channing, Jr.	Boston.
1907	Fuller, Ernest Page	Lawrence.
1907	Gahan, Patrick Francis	Medford.
1906	Gallagher, John Henry Connaughton	Chicopee.
1907	Gallagher, Thomas Morton	Newton.
1906	Galligan, Edward Joseph	Taunton.
1907	George, Arial Wellington	Boston.
1907	Giblin, Francis Joseph	Dorchester.
1906	Giroux, Charles	Three Rivers.
1906	Goldsbury, Paul Williams	Boston.
1907	Goldthwaite, Ralph Harvard	Worcester.
1906	Goodell, William	Springfield.
1906	Green, Robert Montraville	Boston.
1906	Hager, William Perry	Northampton.
1907	Hamilton, Frank Andrew	Somerville.
1906	Hanson, William Thomas	Worcester.
1907	Hatch, Ralph Augustus	Brookline.
1906	Heffner, William James	Northampton.

1906	Henry, Thomas Francis	Worcester.
1907	Hoit, Henry Ambrose	Holden.
1906	Holmes, George Winslow	Boston.
1907	Holt, Charles Herbert	Salem.
1906	Houghton, Richard Henry	East Boston.
1907	Hoyt, Charles Wentworth	Boston.
1906	Hughes, Patrick Joseph	Lawrence.
1907	Jones, Everett	Brookline.
1906	Joyce, Thomas Francis	Lawrence.
1907	Kazanjian, Hampar Paul	Lawrence.
1907	Kent, Bradford	Dorchester.
1907	Kirkpatrick, George Holland	Lynn.
1906	Landers, George Bagnell	Chelsea.
1907	Lawlor, Edward Francis	Lawrence.
1906	Lawlor, Richard Henry	Methuen.
1907	Laws, Sophie Goudge	South Boston.
1906	Leary, Timothy	Roxbury.
1906	Lemieux, Theodule Alfred	Lawrence.
1907	Levy, Felix Julius	Roxbury.
1906	Lloyd, Henry Demarest	Boston.
1906	Lowney, Dennis Joseph	New Bedford.
1907	Lyman, William Robinson	Worthington.
1906	McArdle, John Joseph	Lawrence.
1906	McAvoy, John Joseph	Lawrence.
1907	MacCallum, Wallace Peter	Dorchester.
1907	McCarthy, Louis Flurence	Malden.
1906	McCurdy, Theodore Edward Alexis	Roxbury.
1906	MacKenzie, Lewis Francis	Wellfleet.
1907	Mackinnon, Donald Lauchlin	Boston.
1907	Maloney, Daniel Arthur	Watertown.
1906	Maney, John Joseph	Lawrence.
1907	Masse, John Baptiste	Lawrence.
1907	Mathes, Roy Wentworth	Lynn.
1906	Mehan, Joseph Aloysius	Lowell.
1907	Meyer, Carl Emil	Springfield.
1907	Mignault, Louis Joseph Adolphe	Fall River.
1907	Monahan, John Terence	Fall River.
1907	Morrison, Archibald Benjamin	Boston.
1906	Mullin, Norbert Vincent	Boston.
1907	Myers, Elizabeth Young	Roxbury.
1907	Nevers, Harry Hill	Lawrence.
1907	Norton, Daniel Capron	Salem.
1906	O'Brien, William Smith	Chicopee.
1907	O'Connor, Thomas Hugh	Roxbury.
1907	O'Reilly, James Archer	South Boston.
1907	Packard, Horace	Boston.
1907	Paddock, Brace Whitman	Pittsfield.

1907	Peirce, Bradford Hendrick	Cambridge.
1906	Penhallow, Dunlap Pearce	Boston.
1907	Perkins, Thomas Tounge	Cliftondale.
1906	Petit, Alphonse Herbert	Lawrence.
1907	Phelps, Olney Draper	Worcester.
1907	Phillips, Wilson Frank	Dorchester.
1907	Place, Edwin Hemphill	Boston.
1906	Plunkett, Harold Brabazon	Lowell.
1906	Porter, Fred Knight	Southwick.
1907	Potter, John Claude	South Framingham.
1907	Raymond, Charles Stanley	Northampton.
1907	Redmond, Thomas Henry	Lawrence.
1907	Reid, Isadore Eugene Rosenstein	Jamaica Plain.
1906	Richardson, Horace Kimball	Bradford, Me.
1906	Riordan, Walter Daniel	Lawrence.
1906	Risley, Edward Hammond	Boston.
1901	Rockwell, Alfred Elijah Perkins	Worcester.
1907	Rood, Luther Colby	Dorchester.
1907	Roughan, Charles Michael	Lowell.
1906	Roy, Joseph Napoleon	Webster.
1906	Russell, Frederick James	Newton Centre.
1907	Sawyer, Alpha Reuben	Roslindale.
1907	Schwartz, Myer	Lawrence.
1906	Shanahan, Edward Joseph	Taunton.
1907	Shaw, Adam Ernest	Lowell.
1907	Sherman, Warren Hunter	Westford.
1906	Slutskin, Maurice Louis	Springfield.
1906	Smith, John Hall	Boston.
1906	Smith, Myrtle	Worcester.
1907	Southgate, Alfred Willard	Worcester.
1907	Sparrow, Ernest Harold	Cambridge.
1907	Stammers, John Collins	Charlestown.
1907	Stockbridge, Alberto Horatio	Lynn.
1907	Sullivan, Charles Brent	Hathorne.
1906	Sullivan, John Joseph	Lawrence.
1906	Thayer, Nathaniel Pulsifer	Boston.
1907	Thurber, Madison Templeton	Dorchester.
1906	Tucker, Arthur Wallace	Worcester.
1907	Turner, Charles Sampson	Boston.
1906	Walch, Joseph Francis	Lawrence.
1906	Walcott, William Wright	Natick.
1907	Wardwell, James Knight	Roxbury.
1907	Watters, William Henry	Boston.
1907	Watts, Joseph Palmer	Salem.
1907	Wells, David Washburn	Boston.
1906	Wells, Orion Vassar	Westford.
1906	Wilder, Winford Oliver	Pittsfield.

1906	Williams, John Thomas . .	Boston.
1906	Young, Evangeline Wilson .	Boston.
1907	Young, James Herbert . .	Roxbury.

Total, 171

List of Deceased Fellows.

Admitted.	Name.	Residence.	Date of Death.	Age.
1852	Barrett, William Marshall	Westborough	Nov. 4, 1906	84
1839	Bemis, Charles Vose	Medford	Nov. 6, 1906	90
1874	Bowker, Charles	Bernardston	Dec. 10, 1906	82
1899	Brown, Alphonso Bickford	Newburyport	Oct. 17, 1906	34
1867	Butler, Winthrop	Vineyard Haven	April 23, 1907	68
1900	Carothers, William Harsha	Melrose	Oct. 23, 1906	51
1847	Davis, Robert Thompson	Fall River	Oct. 29, 1906	83
1871	Dearborn, Alvah Burton	Somerville	Aug. 19, 1906	64
1866	Dudley, Henry Watson	Abington	Dec. 29, 1905	75
1862	Everett, James Bradley	Everett	May 20, 1907	78
1875	Finn, James Anthony	Roxbury	Sept. 1, 1906	66
1870	Gaffney, Henry Joseph	Salem	Feb. 9, 1907	59
1881	Hall, Walter Langdon	Medford	Oct. 25, 1906	60
1861	Harlow, John Martyn	Woburn	May 18, 1907	87
1850	Heard, John Theodore	Boston	Sept. 2, 1906	70
1844	Hubbard, Benjamin	Plymouth	Jan. 12, 1907	89
1899	Lecompte, Walter Augustus	Boston	Jan. 13, 1907	36
1892	Lyons, Christopher Philip	Boston	Jan. 22 1907	37
1864	McDougall, Samuel Jay	Boston	Feb. 8, 1907	76
1882	Mackie, George	Attleborough	Aug. 31, 1906	56
1891	McQueeny, Francis Joseph	Boston	Oct. 1, 1906	48
1859	Mansfield, John Robbins	Wakefield	Nov. 11, 1906	74
1841	Mansfield, Joseph D.	Wakefield	May 22, 1907	90
1876	Marion, Otis Humphrey	Allston	Nov. 27, 1906	59
1901	Odiorne, Walter Burlingame	Boston	July 14, 1906	33
1871	Odlin, Charles Cushing	Melrose	Dec. 18, 1906	59
1869	Paun, Amos Bosworth	Middleborough	April 6, 1907	60
1881	Pitcher, Samuel	Hyannis	Feb. 23, 1907	82
1860	Provan, Robert	Brookline	Feb. 23, 1907	73
1906	Roberts, Frederick Albert	Wilmington	Aug. 11, 1906	33
1877	Seymour, James Dwight	Whately	Sept. 10, 1906	56
1884	Spring, Clarence Walter	Fitchburg	Nov. 2, 1906	47
1886	Starbird, Isaac Warren	Roxbury	Feb. 2, 1907	67
1866	Stickney, Alonzo Lawrence	Ashburnham	June 4, 1907	72
1882	Tanner, John Alexander	Dorchester	June 21, 1906	53
1879	Watson, George Henry	Brooklyn	May 25, 1907	59
1896	White, Leonard Darling	Uxbridge	Sept. 18, 1906	47

Total, 37

The Committee On Revision of the By-Laws presented the new draft of By-Laws commended by the Council, and moved that the draft in question, together with the Rules of the Society and the Code of Ethics, be adopted.

Dr. Getchell moved that in

By-Law 10, line 20, after the word "thereof" to insert: "provided no society shall hereby be required to elect a less number of Councillors than chosen for the year 1907."

After discussion by Drs. Harvey, Shattuck and Cabot, the amendment was not adopted, and the motion to adopt the Code as commended by the Council was passed.

On motion of Dr. Harvey it was

Voted, That all By-Laws heretofore in force be repealed.

Voted, That the Secretary be instructed to have printed an edition of the new Code of By-Laws not to exceed 5,000 copies, and that a copy be sent to each Fellow of the Society.

Dr. Cabot presented the following which was adopted:

Resolved, That The Massachusetts Medical Society, having observed, with the highest satisfaction, the great interest which the Governor of this Commonwealth has taken in matters pertaining to Public Health, to which he called the especial attention of the Legislature in his last inaugural address, and with reference to which he has urged, both publicly and privately, the passage of laws to provide for the medical inspection of minors engaged in industrial pursuits, as well as for the suppression of tuberculosis;—and this Society having also perceived the readiness and enthusiasm with which the present Legislature has carried out these suggestions, as shown by the enactment of legislation for the establishment of health districts and the appointment of medical inspectors throughout the State; for the compulsory notification and registration of tuberculosis and for the erection of three sanatoria for the treatment of tuberculosis,—desires to convey to His Excellency Governor Guild, and to the members of the Legislature, its appreciation of their efforts in behalf of the health and welfare of the State, and hereby extends to them a vote of heartfelt thanks.

Resolved, That a copy of these resolutions be transmitted to the Governor of the Commonwealth, to the President of the Senate and to the Speaker of the House of Representatives, and that they be spread upon the records of the Society.

Dr. Harvey stated that several of the schools included in the list of those recognized by the Council are inferior institutions, affording defective instruction to students in medicine. He moved and it was

Voted, That the Committee on Medical Diplomas be instructed to revise the list of Medical Colleges recognized for the purpose set forth in By-Law 1, and report to the next stated meeting of the Council.

Papers were presented as follows:

LEAD POISONING IN A RURAL COMMUNITY.—By Dr. W. N. Cowles, of Ayer.

THE RÖNTGEN RAYS IN PEDIATRICS.—By Dr. T. M. Rotch, of Boston. Remarks by Dr. A. W. George.

At 12 M. the Annual Discourse was given by Dr. J. F. ALLEYNE ADAMS, of Pittsfield.

Voted, That the thanks of the Society be presented to Dr. Adams for his interesting address.

The following delegates from other State Medical Societies were present:

Maine.—Drs. C. D. Smith and R. A. Holland.
New Hampshire.—Dr. R. J. Graves.
Vermont.—Drs. C. B. Ross and C. H. Hazen.

At 1 P.M. the Annual Dinner was served to nearly twelve hundred Fellows.

FRANCIS W. GOSS,
Secretary.

TREASURER'S REPORT.

Receipts.

Balance, April 15, 1906		$11,634 59
Assessments collected by District Treasurers:		
Barnstable	$130 00	
Berkshire	375 00	
Bristol North	275 00	
Bristol South	510 00	
Essex North	430 00	
Essex South	680 00	
Franklin	115 00	
Hampden	780 00	
Hampshire	290 00	
Middlesex East	315 00	
Middlesex North	490 00	
Middlesex South	1,740 00	
Norfolk	1,795 00	
Norfolk South	275 00	
Plymouth	370 00	
Suffolk	3,200 00	
Worcester	1,050 00	
Worcester North	310 00	
	$13,130 00	13,130 00
Assessments, collected at Annual Meeting		770 00
" " by Treasurer		660 00
Sale of Dinner Tickets		18 00
Fees from Exhibitors at Annual Meeting		74 00
Income:		
Interest on deposit New England Trust Co.	$142 74	
Interest on deposit Bay State Trust Co.	128 65	
Interest Franklin Savings Bank	37 93	
" Roxbury Institution for Savings	35 30	
Interest Provident Institution for Savings	35 30	
Interest Suffolk Savings Bank	35 30	
" United States Bond	400 00	
" Massachusetts Bonds	192 50	
" Annuities of Mass. Hospital Life Ins. Co.	816 80	
	$1,824 52	1,824 52
Total		$28,111 11 $28,111 11

Expenses.

President:			
R. R. fares, postage and stationery			$13 00
Secretary:			
Postage and incidentals, two years	$48 26		
Rolls	3 88		
Addressing envelopes	18 25		
Engraving	26 10		
Stenographer	10 00		
Printing	138 50		
	$244 99		244 99
Librarian:			
Index cards	5 50		
Wrappers	17 00		
Printing	26 35		
Delivering Communications	58 39		
Postage	30 00		
	$137 24		137 24
Censors			747 00
Supervisors			20 06
Treasurer:			
Postage stamps	$2 44		
Printing and postage	126 35		
Bay State Trust Co., rent of box	10 00		
Library Bureau, card catalogue	11 25		
Premium on Bond of Treasurer	60 00		
Transportation of accounts	2 00		
Clerks at Annual Meeting	50 00		
	$242 04		242 04
District Treasurers — Commissions	$656 50		
District Treasurers — Postage and printing	166 02		
	$822 52		822 52
Exchange			90
Overpaid assessments, less commission			14 25
Rent			750 00
Salaries			1,400 00
Annual Dividend			4,500 00
Shattuck Lecture			200 00
Cotting Lunch			102 00
Permanent Fund:			
3½ per cent. Massachusetts Bond of 1923	$1,000 00		
Premium	20 00		
Interest	14 97		
	$1,034 97		1,034 97
Amount carried forward,			$10,228 97

Amount brought forward,			$10,228 97	
Committee on By-Laws:				
Printing	46 13			
Typewriting	6 00			
Clerical work	5 00			
Mileage	6 00			
	$63 13		63 13	
Committee on Publications:				
Printing			636 28	
Committee on State and National Legislation:				
Legal services	$350 00			
Stationery	9 00			
	$359 00		359 00	
Annual Meeting:				
Printing, stationery and postage	37 33			
Clerical service	20 50			
Enlarging seal	4 00			
Rent of Mechanics Hall	541 80			
Erecting stand	85 00			
Janitor and coat-room service	13 00			
Use of tables	84 00			
Boston Towel Supply Co.	3 00			
Card signs	12 30			
Transportation	5 50			
Carriages	16 00			
Badges	90			
Police	41 03			
Florist	45 00			
Music	40 00			
Caterer	2,629 41			
Cigars	177 50			
Incidentals	23 25			
	$3,779 52		3,779 52	
Annual Meeting of 1905:				
C. A. Porter, carriage bill	9 76		9 76	
Total			$15,076 66	$15,076 66
Balance, April 15, 1907				$13,034 45
This balance is distributed as follows:				
Balance in New England Trust Co.			$7,017 68	
Deduct out-standing checks			621 09	
Subject to check in New England Trust Co.			$6,396 59	6,396 59
Balance in Bay State Trust Co.				6,593 06
Interest in Suffolk Savings Bank, withdrawn, and deposited in New England Trust Co., April 20				35 50
Cash on hand April 15 and deposited in New England Trust Co., April 17				9 50
				$13,034 45

The permanent fund has been increased by the purchase of one 3½ per cent. bond of the State of Massachusetts, and consists as follows:

Deposit Roxbury Institution for Savings, Cotting Fund	$1,000 00
" Provident Institution for Savings, Cotting Fund	1,000 00
" Suffolk Savings Bank, Cotting Fund . . .	1,000 00
United States 4 per cent. bond, Phillips Fund . .	10,000 00
Annuity bond, Mass. Hospital Life Ins. Co., Shattuck Fund	9,166 87
" " " " " " " . . .	11,253 30
Massachusetts 3½ per cent. bonds	6,000 00
Deposit Franklin Savings Bank	1,074 48
	$40,494 65

EDWARD M. BUCKINGHAM,
Treasurer.

The undersigned, a duly appointed committee, having examined the books of the treasurer, find them correctly cast and properly vouched, and also that he has in his possession the securities called for.

EDWIN ALLEN LOCKE, *Chairman.*
M. V. PIERCE.

Boston, May 21, 1907.

Officers of The Massachusetts Medical Society.

1907—1908.

Chosen June 11, 1907.

George W. Gay,	Newton,	President.
Francis J. Canedy,	Shelburne Falls,	Vice-President.
Francis W. Goss,	Roxbury,	Secretary.
Edward M. Buckingham,	Boston,	Treasurer.
Edwin H. Brigham,	Brookline,	Librarian.

Standing Committees.

Of Arrangements.

G. C. S. Badger, C. C. Simmons,
Lincoln Davis, G. H. Francis,
L. W. Gilbert, A. N. Broughton.

On Publications.

O. F. Wadsworth, G. B. Shattuck, H. L. Burrell.

On Membership and Finance.

F. W. Goss, Walter Ela, C. M. Green,
A. Coolidge, Jr., H. A. Wood.

To Procure Scientific Papers.

B. P. Croft, R. C. Larrabee, M. D. Clarke,
Charles Harrington, R. B. Greenough.

On Ethics and Discipline.

C. G. Carleton, J. F. A. Adams, Leonard Wheeler,
J. A. Gage, J. W. Bartol.

On Medical Diplomas.

H. E. Marion, O. F. Rogers, H. W. Newhall.

On Medical Education.

H. C. Ernst, H. D. Arnold, C. H. Williams.

On State and National Legislation.

G. W. Gay, S. D. Presbrey, G. G. Sears,
D. D. Gilbert, C. H. Cook.

Presidents of District Societies—Vice-Presidents (Ex-Officiis).

[Arranged according to seniority.]

C. P. Putnam,	N. S. Hunting,
T. M. Durell,	W. J. Sullivan,
W. A. Johnson,	C. W. Harlow,
E. V. Scribner,	J. E. Simpson,
A. E. Mossman,	J. W. Cram,
F. G. Wheatley,	C. A. Allen,
G. deN. Hough,	E. Washburn,
Samuel Crowell,	G. L. Rice,
J. G. Hayes,	C. M. Baker.

Councillors.

Barnstable.—R. H. Faunce, Sandwich; H. B. Hart, East Dennis.

Berkshire.—J. F. A. Adams, Pittsfield; O. J. Brown, H. Bushnell, North Adams; H. Colt, I. S. F. Dodd, Pittsfield; A. T. Wakefield, Sheffield.

Bristol North.—A. S. Deane, F. A. Hubbard, S. D. Presbrey, T. J. Robinson, Taunton; A. M. Round, Norton.

Bristol South.—J. A. Barré, A. W. Buck, W. A. Dolan, J. H. Gifford, H. G. Wilbur, Fall River; C. A. Bonney, Jr., H. C. Kirby, C. A. Pratt, J. C. Pothier, New Bedford.

Essex North.—J. J. Bartley, J. F. Burnham, Lawrence; I. J. Clarke, Haverhill; J. A. Douglass, Amesbury; J. A. Hogan, Lawrence; A. H. Peirce, West Newbury; W. W. Pillsbury, Newburyport; J. W. Rand, Amesbury; L. J. Young, Haverhill.

Essex South.—C. H. Bangs, Lynn; H. C. Boutelle, Danvers; S. P. F. Cooke, Gloucester; H. K. Foster, Peabody;

H. J. Hall, Marblehead; P. P. Johnson, Beverly; E. W. Jones, W. B. Little, C. E. Meader, J. F. O'Shea, Lynn; H. W. Mitchell, Hathorne; G. E. MacArthur, Ipswich; E. L. Peirson, Salem; P. C. Proctor, Gloucester; H. E. Sears, J. H. Shatswell, Beverly.

FRANKLIN.—H. G. Stetson, G. P. Twitchell, Greenfield; N. P. Wood, Northfield.

HAMPDEN.—J. B. Atwater, Westfield; L. S. Brooks, F. W. Chapin, E. H. Guild, Springfield; E. Flood, Palmer; J. W. Hannum, Ludlow; J. C. Hubbard, S. A. Mahoney, Holyoke; C. W. Jackson, Monson; F. F. Parker, Chicopee; W. S. Pomeroy, A. O. Squier, W. R. Weiser, Springfield; H. G. Webber, Wilbraham.

HAMPSHIRE.—A. M. Belden, Northampton; O. N. Cobb, Easthampton; N. Holden, Southampton; V. S. Merritt, Haydenville; F. H. Smith, Hadley.

MIDDLESEX EAST.—H. G. Blake, Woburn; G. F. Dow, Reading; J. W. Heath, Wakefield; W. H. Keleher, Woburn; R. D. Perley, Melrose.

MIDDLESEX NORTH.—J. B. Field, W. M. Jones, J. E. Lamoureux, W. P. Lawler, J. B. O'Connor, C. E. Simpson, E. J. Welch, Lowell; J. H. Nichols, Tewksbury; W. J. Sleeper, Westford; F. E. Varney, North Chelmsford.

MIDDLESEX SOUTH.—T. Chamberlin, Concord; N. F. Chandler, Medford; J. M. Crocker, Cambridge; T. M. Durell, D. S. Flewelling, Somerville; G. W. Gay, *President*, Chestnut Hill; W. Ela, J. L. Hildreth, Cambridge; E. G. Hoitt, Marlboro'; A. A. Jackson, Everett; C. D. McCarthy, Malden; J. A. McDonald, Charlestown; H. E. Marion, Brighton; J. A Mead, Watertown; G. A. Miles, Somerville; J. T. G. Nichols, W. Preble, Cambridge; L. M. Palmer, South Framingham; C. E. Prior, G. Ryder, Malden; H. S. Rowen, Brighton; C. D. Sawin, Charlestown; F. M. Sherman, L. R. Stone, Newton; F. G. Smith, Somerville; I. E. Stowe, Medford; J. E. Somers, E. H. Stevens, F. W. Taylor, H. P. Walcott, Cambridge; W. H. Sylvester, Natick; J. Tolman, Arlington; A. M. Wilkins, South Framingham; H. A. Wood, A. Worcester, Waltham.

NORFOLK.—D. N. Blakely, Roxbury; B. S. Blanchard, H. L. Chase, F. W. Draper, G. H. Francis, Brookline; M. J. Cronin, C. B. Darling, T. F. Greene, Roxbury; S. W. Crittenden, C. G. Dewey, D. D. Gilbert, J. S. Greene, Dorchester; H. R. Hitchcock, Hyde Park; H. T. Holland, Jamaica Plain; W. W. Howell, West Roxbury; G. N. Jones, Wellesley Hills; A. A. Macdonald, E. G. Morse, J. C. D. Pigeon, Roxbury; H. T. Mansfield, Needham; M. V. Pierce, Milton; J. W. Pratt, Dedham; O. F. Rogers, Dorchester; W. H. Robinson, T. Smith, Jamaica Plain; H. R. Stedman, Brookline; H. B. Stevens, West Roxbury; H. C. Towle, J. P. Treanor, E. T. Twitchell, Dorchester; E. Thayer, W. A. White, C. H. Winn, Roxbury.

NORFOLK SOUTH.—T. J. Dion, West Quincy; R. McLellan, Quincy; W. H. W. Sturgis, Allerton; V. M. Tirrell, South Weymouth.

PLYMOUTH.—J. H. Averill, Campello; C. W. Bartlett, Marshfield; N. K. Noyes, Duxbury; G. Osgood, Rockland; A. E. Paine, Brockton; F. G. Wheatley, North Abington.

SUFFOLK.—H. D. Arnold, J. B. Ayer, F. G. Balch, E. G. Brackett, E. H. Bradford, E. M. Buckingham, *Treasurer*, H. L. Burrell, J. F. Bush, A. T. Cabot, A. Coolidge, Jr., F. J. Cotton, W. T. Councilman, E. G. Cutler, H. Derby, R. H. Fitz, C. F. Folsom, W. W. Gannett, C. M. Green, F. B. Harrington, H. Jackson, B. J. Jeffries, G. F. Jelly, E. P. Joslin, F. I. Knight, H. A. Lothrop, A. L. Mason, J. H. McCollom, A. B. Morong, J. G. Mumford, J. C. Munro, A. H. Nichols, C. B. Porter, A. Post, C. P. Putnam, J. J. Putnan, W. L. Richardson, T. M. Rotch, G. H. M. Rowe, J. D. K. Sabine, G. G. Sears, F. C. Shattuck, G. B. Shattuck, A. K. Stone, J. B. Swift, H. F. Vickery, O. F. Wadsworth, J. C. Warren, G. H. Washburn, J. C. White, H. Williams, G. Wolcott, Boston; E. S. Boland, M. F. Gavin, South Boston; J. M. Putnam, Chelsea.

WORCESTER.—W. P. Bowers, Clinton; P. P. Comey, J. T. Duggan, O. H. Everett, G. E. Francis, H. Gage, T. H. Gage, A. C. Getchell, R. W. Greene, D. Harrower, Worcester; E. B. Harvey, Westboro'; S. P. Holbrook, East Douglas; E. W. Norwood, Spencer; W. G. Reed, Southbridge; H. M. Quinby, E. V. Scribner, L. Wheeler, A. Wood, L. F. Woodward, S. B. Woodward, Worcester.

WORCESTER NORTH.—C. E. Bigelow, Leominster; S. E. Greenwood, Templeton; H. H. Lyons, E. P. Miller, F. H. Thompson, Fitchburg; E. A. Sawyer, Gardner.

Censors.

BARNSTABLE.—S. T. Davis, Orleans; R. H. Faunce, *Supervisor*, Sandwich; S. F. Haskins, Cotuit; J. H. Higgins, Marston's Mills; E. M. Parker, South Yarmouth.

BERKSHIRE.—J. F. A. Adams, *Supervisor*, Pittsfield; M. M. Brown, H. Bushnell, North Adams; W. M. Mercer, Pittsfield; W. W Scofield, Dalton.

BRISTOL NORTH.—A. R. Crandell, F. A. Hubbard, *Supervisor*, D. J. Mehegan, T. J. Robinson, Taunton; H. S. Kilby, North Attleborough.

BRISTOL SOUTH.—S. W. Bowen, A. I. Connell, W. A. Dolan, *Supervisor*, Fall River; C. F. Connor, A. P. Webber, New Bedford.

ESSEX NORTH.—R. M. Birmingham, Lawrence; J. A. Fitz-Hugh, Amesbury; F. E. Sweetsir, Merrimac; F. B. Pierce, L. J. Young, *Supervisor*, Haverhill.

ESSEX SOUTH.—F. S. Atwood, Salem; F. W. Baldwin, Danvers; M. A. Harmon, H. W. Newhall, Lynn; H. E. Sears, *Supervisor*, Beverly.

FRANKLIN.—E. G. Best, Turner's Falls; G. A. Cooke, Montague; G. R. Fessenden, Ashfield; C. G. Trow, Sunderland; N. P. Wood, *Supervisor*, Northfield.

HAMPDEN.—A. L. Damon, North Wilbraham; D. E. Keefe, J. E. Marsh, F. B. Sweet, W. R. Weiser, *Supervisor*, Springfield.

HAMPSHIRE.—W. H. Adams, A. M. Belden, *Supervisor*, J. C. Fahey, Northampton; N. C. Haskell, Amherst; J. Stowell, North Amherst.

MIDDLESEX EAST.—C. F. Dow, *Supervisor*, E. D. Richmond, Reading; E. C. Fish, Melrose; C. F. McCarthy, Winchester; M. D. Sheehan, Stoneham.

MIDDLESEX NORTH.—T. G. McGannon, J. V. Meigs, G. E. Pinkham, O. P. Porter, C. E. Simpson, *Supervisor*, Lowell.

MIDDLESEX SOUTH.—G. A. Bancroft, South Natick; H. D. Chadwick, Waltham; L. M. Palmer, *Supervisor*, South Framingham; E. D. Pillsbury, Somerville; H. S. Rowen, Brighton.

NORFOLK.—C. S. Francis, Brookline; E. T. Galligan, Roxbury; G. H. Ingalls, Jamaica Plain; R. M. Merrick, E. T. Twitchell, *Supervisor*, Dorchester.

NORFOLK SOUTH.—J. H. Ash, West Quincy; D. A. Bruce, Atlantic; G. D. Bullock, Weymouth; J. H. Libbey, East Weymouth; V. M. Tirrell, *Supervisor*, South Weymouth.

PLYMOUTH.—J. E. Bacon, W. P. Chisholm, Brockton; N. C. King, Campello; A. A. MacKeen, Whitman; G. Osgood, *Supervisor*, Rockland.

SUFFOLK.—J. N. Coolidge, W. E. Fay, W. F. Gay, C. H. Hare, A. K. Stone, *Supervisor*, Boston.

WORCESTER.—F. H. Baker, W. T. Clark, E. V. Scribner, *Supervisor*, R. P. Watkins, Worcester; F. H. Clapp, North Grafton.

WORCESTER NORTH.—J. B. Donnelly, West Gardner; E. P. Miller, *Supervisor*, E. J. Tully, Fitchburg; H. W. Page, Baldwinsville; A. H. Pierce, Leominster.

Commissioners of Trials.

BARNSTABLE	E. S. Osborne	West Dennis.
BERKSHIRE	A. T. Wakefield	Sheffield.
BRISTOL NORTH	C. S. Holden	Attleborough.
BRISTOL SOUTH	G. L. Richards	Fall River.
ESSEX NORTH	J. F. Young	Newburyport.
ESSEX SOUTH	J. Allen	Topsfield.
FRANKLIN	F. E. Johnson	Erving.
HAMPDEN	W. A. Smith	Springfield.
HAMPSHIRE	C. Seymour	Northampton.
MIDDLESEX EAST	S. W. Kelley	Woburn.
MIDDLESEX NORTH	G. A. Harlow	Tyngsboro'.
MIDDLESEX SOUTH	H. P. Makechnie	West Somerville.

NORFOLK	M. V. Pierce	Milton.
NORFOLK SOUTH	G. W. Tinkham	Weymouth.
PLYMOUTH	C. A. Drew	State Farm.
SUFFOLK	M. F. Gavin	Boston.
WORCESTER	C. H. Perry	Worcester.
WORCESTER NORTH	L. G. Chandler	Townsend.

Officers of the District Medical Societies.

BARNSTABLE.—C. M. Baker, Hyannis, *President;* S. H. Sears, Yarmouthport, *Vice-President;* J. P. Nickerson, West Harwich, *Secretary;* W. D. Kinney, Osterville, *Treasurer.*

BERKSHIRE.—G. L. Rice, North Adams, *President;* J. J. Hassett, Lee, *Vice-President;* I. S. F. Dodd, Pittsfield, *Secretary;* W. L. Paddock, Pittsfield, *Treasurer.*

BRISTOL NORTH.—E. Washburn, Taunton, *President;* A. V. Goss, Taunton, *Vice-President;* R. D. Dean, Taunton, *Secretary;* W. Y. Fox, Taunton, *Treasurer.*

BRISTOL SOUTH.—G. deN. Hough, New Bedford, *President;* W. T. Learned, Fall River, *Vice-President;* A. J. Abbe, Fall River, *Secretary and Treasurer.*

ESSEX NORTH.—W. J. Sullivan, Lawrence, *President;* C. E. Durant, Haverhill, *Vice-President;* M. D. Clarke, Haverhill, *Secretary and Treasurer.*

ESSEX SOUTH.—J. E. Simpson, Salem, *President;* F. E. Stone, Lynn, *Vice-President;* G. K. Blair, Salem, *Secretary;* G. Z. Goodell, Salem, *Treasurer.*

FRANKLIN.—J. W. Cram, Colrain, *President;* C. L. Upton, Shelburne Falls, *Vice-President;* C. M. Greenough, Greenfield, *Secretary and Treasurer.*

HAMPDEN.—C. A. Allen, Holyoke, *President;* G. L. Woods, Springfield, *Vice-President;* T. S. Bacon, Springfield, *Secretary and Treasurer.*

HAMPSHIRE.—J. G. Hayes, Williamsburg, *President;* G. D. Thayer, Northampton, *Vice-President;* A. G. Minshall, Northampton, *Secretary;* A. H. Hoadley, Northampton, *Treasurer.*

MIDDLESEX EAST.—C. W. Harlow, Melrose Highlands, *President;* F. E. Park, Stoneham, *Vice-President;* H. A. Gale, Winchester, *Secretary;* C. Dutton, Wakefield, *Treasurer.*

MIDDLESEX NORTH.—W. A. Johnson, Lowell, *President;* W. B. Jackson, Lowell, *Vice-President;* R. J. Meigs, Lowell, *Secretary;* T. B. Smith, Lowell, *Treasurer.*

MIDDLESEX SOUTH.—T. M. Durrell, Somerville, *President;* C. E. Prior, Malden, *Vice-President;* F. R. Jouett, Cambridge, *Secretary;* W. Ela, Cambridge, *Treasurer.*

NORFOLK.—S. Crowell, Dorchester, *President;* A. P. Perry, Jamaica Plain, *Vice-President;* W. H. Robey, Jr., Roxbury, *Secretary;* E. G. Morse, Roxbury, *Treasurer.*

NORFOLK SOUTH.—N. S. Hunting, Quincy, *President;* O. H. Howe, Cohasset, *Vice-President;* C. S. Adams, Wollaston, *Secretary and Treasurer.*

PLYMOUTH.—F. G. Wheatley, North Abington, *President;* F. J. Ripley, Brockton, *Vice-President;* A. C. Smith, Brockton, *Secretary and Treasurer.*

SUFFOLK.—C. P. Putnam, Boston, *President;* G. H. Monks, Boston, *Vice-President;* L. R. G. Crandon, Boston, *Secretary;* W. H. Prescott, Boston, *Treasurer.*

WORCESTER.—E. V. Scribner, Worcester, *President;* W. G. Reed, Southbridge, *Vice-President;* G. E. Emery, Worcester, *Secretary;* G. O. Ward, Worcester, *Treasurer.*

WORCESTER NORTH.—A. E. Mossman, Westminster, *President;* J. G. Henry, Winchendon, *Vice-President;* W. F. Sawyer, Fitchburg, *Secretary;* E. L. Fiske, Fitchburg, *Treasurer.*

SUPPLEMENT No. I. TO CATALOGUE OF 1905.

Admissions reported since January 1, 1905.

1905	Abbott, Harry Daniel	Danvers.
1905	Ash, Thomas Francis	Rockland.
1905	Bailey, Frederick James	Dorchester.
1905	Barney, James Dellinger	Boston.
1905	Bastian, George Leon	Lowell.
1905	Bliss, George Stephen	Waverley.
1905	Bonoff, Zelly Adam	Worcester.
1905	Boutwell, Horace Keith	Boston.
1905	Burke, Francis Ramon	Quincy.
1905	Burnham, Parker	Gloucester.
1905	Butler, Patrick Francis	South Boston.
1905	Chandler, Clarence Luther	Fitchburg.
1905	Chapman, Howard Jones	Everett.
1905	Chesley, Alfred Ervan	Lawrence.
1905	Clark, George Oliver	Boston.
1905	Cook, Philip Howard	Worcester.
1905	Cooley, Abbott Lathrop	Chicopee Falls.
1905	Coulson, Richard	Somerville.
1905	Daly, Jeremiah James	North Andover.
1905	Dexter, Fred Fay	Granby.
1905	Easton, Charles Daniel	Boston.
1905	Everett, Frederick Luther	Springfield.
1905	Fitch, Ralph Roswell	Dorchester.
1905	Flett, Penelope McNaughton	Waverley.
1905	Floyd, Cleaveland	Boston.
1905	Freedman, Louis Mark	Chelsea.
1905	Gafney, Harry Dabol	Ware.
1905	Garland, Roy	Boston.
1905	Gay, Clarence Bertram	Fitchburg.
1905	Greenwood, Arthur Moses	Boston.
1905	Hamilton, Robert De Lancey	Newburyport.
1905	Harper, Alfred Alphonso	North Adams.
1905	Hartwell, John Bryant	Boston.
1905	Hayes, Justin Edward	Chesterfield.

1905	Hedin, Carl Johan	Tewksbury.
1905	Hussey, Edward John	Boston.
1905	Jackson, John Patrick	Fall River.
1905	Kelly, William Dugan	Boston.
1905	Kenney, Walter Clement	Tewksbury.
1905	Kent, Ralph Porter	Attleboro'.
1905	Kimpton, Arthur Ronald	Somerville.
1905	Larrabee, Herbert Manson	Tewksbury.
1905	Lee, William George	Boston.
1905	Linenthal, Harry	Roxbury.
1905	MacAusland, William Russell	Boston.
1905	McCaffrey, Charles Francis	Somerville.
1905	MacCormick, John Allan	Boston.
1905	Mackillop, Daniel	Cambridge.
1905	McPherson, George Edwin	South Boston.
1905	MacQuaid, John Stephen	Springfield.
1905	Makechnie, Arthur North	Cambridge.
1905	Mayberry, Frank Eugene	Boston.
1905	Meehan, Patrick Joseph	Lowell.
1905	Mellus, Edward	Worcester.
1905	Mendelsohn, Louis	Dorchester.
1905	Messinger, Harry Carleton	South Boston.
1905	Murphy, Thomas William	Lawrence.
1905	Myers, Edward Everett	Roxbury.
1905	O'Brien, Loretta Joy	Boston.
1905	Osgood, George	Boston.
1905	Parr, John	Methuen.
1905	Patterson, Alice Maria	Peabody.
1905	Pike, Forrest Fay	Melrose.
1905	Powers, George Herman, Jr.	Boston.
1905	Quessy, Alfred Henry	Fitchburg.
1905	Reardon, Daniel Bartholomew	Quincy.
1905	Rumrill, Samuel Dudley	Springfield.
1905	Sargent, Walter Leslie	Quincy.
1905	Scanlan, Thomas John	Boston.
1905	Segur, Willard Blossom	Enfield.
1905	Seymour, Frederick Ward	Boston.
1905	Sheehan, William Joseph	South Boston.
1905	Simmons, Fred Albert	Brockton.
1905	Sinclitico, Guiseppe	Lawrence.
1905	Stoneman, Edgar Amos	Springfield.
1905	Topaz, Anna	Boston.
1905	Vrooman, Earle Morey	Cummington.
1905	Wallace, Anna Marie	Waverley.
1905	Whitney, Edward William	Ware.
1905	Whitney, Harvey Herbert	Holyoke.
1905	Woodbury, Willard Porter	Beverly.

List of Deceased Fellows.

Admitted.	Name.	Residence.	Date of Death.	Age.
1880	Allen, Franklin Haley	Haverhill	April 23, 1905	49
1899	Brown, Anthony Leopold	Springfield	April 22, 1905	42
1902	Brown, Henry Rienzi	Leominster	Feb. 16, 1905	63
1900	Creesy, Everett Larcom	Beverly	May 28, 1905	30
1897	Donlan, John Malachi	Fitchburg	Jan'y 26, 1905	30
1894	Drown, Edward Payson	Malden	May 25, 1905	39
1870	Hunt, Israel Thorndike	Charlestown	Feb. 16, 1905	63
1848	Hunt, Otis Eugene	Newton	Jan'y 20, 1905	82
1890	King, William Rufus	Boston	Feb. 17, 1905	37
1888	Lombard, John Patrick	Dorchester	March 21, 1905	44
1864	Newhall, Edward	Lynn	June 12, 1905	83
1890	Padula, Thomas Francis	Quincy	Feb. 9, 1905	45
1875	Phelps, Rollin Horace	Littleton	Jan'y 28, 1905	71
1890	Swift, Lawrence Chew	Pittsfield	June 1, 1905	53
1871	Thayer, Samuel Ezra	Wenham	Feb. 5, 1905	62
1887	Warren, William Barnard	Groton	April 29, 1905	51

The following names have been changed:

Purvis, Alice Hatheway to Robie, Alice Hatheway.

Young, Emma Elizabeth to Young-Slaughter, Emma Elizabeth.

The following have been placed on the retired list:

Winthrop Butler, of Vineyard Haven.
George W. Doane, of Hyannis.
Willard S. Everett, of Waban.
James Hedenberg, of Medford.
William H. Lathrop, of Lowell.
Charles P. Morrill, of North Andover.
William Neilson, of Marblehead.
Samuel Pitcher, of Hyannis.

The following resignations have been accepted:

Harrie S. Baketel, of Jersey City, N. J.
Noah A. Burr, of South Manchester, Conn.
Allston F. Hunt, of Swampscott.
Charles H. Kingsbury, of Danielson, Conn.
Charles F. Morse, of U. S. Army.
Mary E. Newcomb, of Blissfield, Mich.
Richard R. Pettigrew, of Scranton, Pa.
Thomas G. Reed, of South Boston.
Walter N. Sharp, of Indianapolis, Ind.

The following have been expelled:

Howard K. Glidden, of Lynn.
Seraph Frissell, of Springfield.
Charles A. Eastman, of Old Orchard, Me.

SUPPLEMENT No. II. TO CATALOGUE OF 1905.

Admissions reported from June 13, 1905, to June 12, 1906.

1906	Adams, Charles Waldron	Cambridge.
1906	Arkin, Louis	Roxbury.
1905	Arms, Burdett Loomis	Brookline.
1905	Ascher, Joseph	Boston.
1905	Atwater, James Billings	Westfield.
1906	Austin, Albert Elmer	Medway.
1906	Balboni, Gerardo Monari	Boston.
1905	Bardwell, Frederick Albert	Roxbury.
1906	Barnes, William Lester	Lexington.
1905	Bartlett, Walter Oscar	Roxbury.
1906	Bartol, Edward Francis Washburn	Milton.
1906	Beach, Sylvester Judd	Boston.
1905	Beckett, Francis Henry	Fall River.
1906	Bennett, Hamlin Perley	Woodstock, Vt.
1905	Bianco, Joseph Anthony	East Boston.
1906	Bigelow, Alice Houghton	Jamaica Plain.
1905	Bigelow, Edward Bridge	Worcester.
1906	Blake, Gerald	Boston.
1906	Boardman, William Parsons	Boston.
1906	Borden, George Edward	Fall River.
1906	Bosworth, Freeman Dodd	Tewksbury.
1906	Brady, Frank Robert	Lowell.
1905	Brennan, Thomas Joseph	New Bedford.
1906	Brindisi, Rocco	Boston.
1906	Brooks Edith May	Roxbury.
1905	Brown, Edward Manning	Springfield.
1906	Brown, Wallace Everett	North Adams.
1906	Bryant, Clarence Edmund	Cambridge.
1906	Buckley, William Stephen	Brighton.
1906	Burnett, Francis Lowell	Cambridge.
1906	Bushnell, Homer Andrew	North Adams.
1905	Butler, Joel Ives	Springfield.
1906	Carney, Patrick Joseph	Dorchester.
1906	Carr, Percy Whitman	Hyde Park.

1905	Carroll, John Joseph	Holyoke.
1905	Carvill, Lizzie Maud	Somerville.
1905	Cavanaugh, Thomas Edward	Holyoke.
1905	Celce, Frank Frederick	Holyoke.
1905	Celce, Jean Henriette	Holyoke.
1906	Chisholm, Miles Dudley	Westfield.
1906	Churchill, John Darling	Plymouth.
1906	Clark, John Donovan	Auburndale.
1905	Clark, Nelson Henry	Boston.
1905	Cockett, Marguerite Standish	Roxbury.
1905	Coffrey, William Francis	Westfield.
1905	Coleman, David Charles	Beverly.
1905	Collins, Aubrey John	Mattapan.
1906	Cooper, Alden Vernon	Palmer.
1905	Corey, Frederick Hall	Roxbury.
1906	Costello, John Henry	Dorchester.
1905	Coté, Honoré Joseph	Boston.
1905	Cox, Simon Francis	Boston.
1905	Craig, William Gibson	Springfield.
1906	Crane, Bayard Taylor	Rutland.
1905	Cregg, Francis Aloysius	Lowell.
1905	Curley, Clarence Proctor	Provincetown.
1905	Curran, Simon Francis	Dorchester.
1906	Cushing, Arthur Alden	Brookline.
1905	Dacey, Cornelius Joseph	Brockton.
1906	Dana, Harold Ward	Boston.
1906	Danforth, Harland Abbott	Lynn.
1906	Davidson, Henry James Duff	Springfield.
1906	Davis, Charles Augustus	Palmer.
1906	Davis, Minot Flagg	Boston.
1905	Davison, Arthur Howard	Dorchester.
1905	Day, Hilbert Francis	Boston.
1906	Dearborn, Henry Hale	Leominster.
1905	Deering, George Edwin	Worcester.
1906	Denning, Edward John	South Boston.
1906	Dexter, Richard	Boston.
1905	Dore, Francis James	Roxbury.
1905	Douglas, Archibald John	Westfield.
1906	Dubois, Eoline Church	Cambridge.
1906	Duffee, Thomas Edward	Winchendon.
1905	Dugdale, Frederick	Lowell.
1906	Dunbar, Francis Herbert	Boston.
1905	Duryee, Albert Plummer	Brockton.
1906	Dutcher, William Austin	Boston.
1906	Dutton, Julius Maltby	Westfield.
1905	Dwyer, William Joseph	Cambridge.
1906	Eastman, Theodore Jewett	Boston.

1906	Ehrenfried, Albert	Boston.
1906	Eldredge, Harvey Loud	East Boston.
1906	Ellam, Herbert William	Worcester.
1905	Ellis, Edward Keith	Boston.
1905	Emmons, Arthur Brewster	Brookline.
1906	Fabyan, Marshal	Brookline.
1906	Farmer, William Francis	Holden.
1905	Faunce, Calvin Barstow, Jr.	Jamaica Plain.
1905	Faxon, Dora Winifred	Taunton.
1905	Fenwick, George Benson	Worcester.
1906	Field, Henry Martyn	Dorchester.
1906	Fitzgerald, John Joseph	Haverhill.
1905	Flynn, Henry Lawrence	Roxbury.
1905	Furrer, Arnold Frotcham	Cleveland, O.
1905	Gabler, George Lewis	Holyoke.
1905	Gage, Fred Leon	Worcester.
1905	Gates, George Cushman	Chicopee.
1906	Gile, Frank Herbert, Jr.	Melrose Highlands.
1906	Gill, Mary Eva	Dorchester.
1905	Gillis, John Ewen	West Somerville.
1906	Gilman, Charles Sleeper	Boston.
1906	Gilpatrick, Roy Hawkes	Boston.
1906	Glass, James	South Framingham.
1906	Goddard, Samuel Warren	Brockton.
1905	Goddu, Louis Adalore Oliver	Beverly.
1906	Good, Frederick Leo	Boston.
1905	Gorham, George Hartley	Tabernilla, C. Z.
1905	Graves, Benjamin Augustus	Dorchester.
1906	Graves, James Chapman, Jr.	Boston.
1905	Haché, Henry Clement	Somerville.
1906	Halligan, Edward Maurice	South Boston.
1906	Halsall, Mary Elizabeth	East Boston.
1906	Hammond, Charles	Hanover.
1906	Hancock, Albert William	Lawrence.
1905	Hanson, William Clinton	Cambridge.
1906	Hardwick, Sydney Curtis	Quincy.
1905	Harrington, Robert Brine	Northampton.
1906	Harrison, Henry	Boston.
1906	Hartman, Gustave	Lynn.
1906	Hartnett, Edward Daniel	East Boston.
1906	Hatch, Royal	Boston
1906	Heffernan, David Aloysius	Boston.
1906	Hicks, George Henry	Fall River.
1905	Hinchliffe, Frederick	Lowell.
1906	Hoitt, Charles Lewis	Lynn.
1906	Holt, Harry Frye	Worcester.
1905	Hooker, Stuart VanRensselaer	Boston.

1906	Hopkinson, George	Boston.
1905	Hornbrook, Francis Wheeler	Newton.
1906	Horne, Lester Wallace	Acushnet.
1906	Hosley, Walter Alexis	Boston.
1905	Hunt, Alice Elizabeth Palmer	Holyoke.
1905	Hunter, Norman McLeod	Hudson.
1906	Hurley, Edward Daniel	South Boston.
1906	Janes, Arthur Percy	Melrose Highlands.
1905	Jennings, Curtis Hermon	Fitchburg.
1905	Jones, Frederick Elmer	Roxbury.
1905	Jones, Wellington West	Housatonic.
1905	Jordan, John Franklin	Peabody.
1906	Keeler, William Basil	Roxbury.
1905	Keeley, Ambrose Augustine	Fall River.
1905	Kennedy, Edward Anthony	Monson.
1906	Kidner, Frederick Clinton	Boston.
1906	Kingsbury, Walter Warren	Malden.
1905	Kingsley, Patrick Joseph	Dorchester.
1906	Lahey, Francis Howard	Boston.
1905	Lane, John William	Boston.
1906	Law, Katharine Hayes	Boston.
1905	Leary, Chrysostom John	New Bedford.
1905	Le Gro, Lester Burnside	Haverhill.
1906	Lewis, Elisha Sears	Princeton.
1906	Lewis, James Prince	Boston.
1906	Long, Merritt Allen	Manchester.
1905	Longfellow, Henry Nathan	Roxbury.
1905	Lowell, William Holbrook	Boston.
1905	Lowenstein, Fred Philip	Westfield.
1905	Lucas, Julian Dyer	Roxbury.
1906	McAllester, John Joseph Hector	New Bedford.
1905	McAllester, Ralph William	Everett.
1906	McCarthy, Francis Patrick	Jamaica Plain.
1906	McCarthy, Timothy Francis	East Boston.
1905	McCauley, James Douthard	Cambridge.
1906	McCormick, John Joseph	South Boston.
1906	Macdonald, Frederick Cornelius	Roxbury.
1906	MacKerrow, Horace Gilford	Worcester.
1906	MacOdrum, Angus	Boston.
1905	McSheehy, Theobald Coleman	Worcester.
1905	Magrath, George Burgess	Boston.
1906	Maguire, Eugene Leo	South Boston.
1905	Mahoney, Francis Xavier	Dorchester.
1905	Maloney, Thomas Aloysius	Springfield.
1905	Marshall, Harold Kenneth	Dorchester.
1906	Mather, John Adams	Colrain.
1906	Means, Philip Corydon	Chelsea.

1905	Medalia, Leon Samuel	Boston.
1905	Merrill, Ayres Phillip	Pittsfield.
1906	Miller, Albert Eber	Needham.
1906	Miller, Malcolm Dean	Boston.
1906	Miller, Vesta Delphene	Needham.
1906	Mills, Alvah Vernon	Brookline.
1906	Miner, Harold Edson	Holyoke.
1906	Mitchell, Elmer Wesley	Fall River.
1906	Morrow, William Robert	South Framingham.
1905	Morse, Nathaniel Niles	New York, N. Y.
1905	Morse, Vernon Harcourt Chipman	Cambridge.
1906	Mullin, Estella Loretta	Worcester.
1905	Mullins, Eugene Norton	Baldwinsville.
1906	Munroe, Harrington Bennett	Lynn.
1905	Murphy, Frederick Paul	Lowell.
1905	Murphy, Frederick Vincent	Attleborough.
1905	Nagle, Evelyn Wyman	Boston.
1905	Noyes, Margaret Louise	Boston.
1905	Noyes, William Nelson	Fitchburg.
1906	Oak, Charles Arthur	Revere.
1905	O'Brien, Daniel Paul	New Bedford.
1905	O'Brien, Joseph Jeremiah	Dorchester.
1905	O'Connell, Andrew Edward	Worcester.
1906	O'Connor, John Christopher	Salem.
1906	Oliver, Everard Lawrence	Boston.
1905	O'Malley, Edward Francis	Fitchburg.
1905	Ordway, Mabel Dyer	Boston.
1905	Ordway, Thomas	Boston.
1906	Orton, Samuel Torrey	Boston.
1905	O'Sullivan, Francis Michael Aloysius	Lowell.
1905	Paine, Alonzo Kingman	Boston.
1905	Paquin, Joseph Ubalde	New Bedford.
1906	Parker, Albert Munro	Boston.
1906	Pascoe, William Whelan	Savoy.
1905	Pattrell, Arthur Ellis	North Grafton.
1906	Pease, Lewis Waite	Hyannis.
1906	Potter, Philip Sheridan	North Adams.
1905	Powers, Herbert Hale	Brookline.
1905	Prescott, Henry Dudley	New Bedford.
1906	Purvis, Charles Burleigh	Boston.
1906	Reynolds, John Timothy	Quincy.
1905	Rice, Alexander Hamilton	Boston.
1906	Rice, Allen Galpin	Boston.
1906	Riley, John Henry	North Adams.
1906	Roach, Alfred John	Lowell.
1906	Robbins, Chandler	Boston.
1906	Robbins, William Bradford	Boston.

1906	Roberts, Frederick Albert	Wilmington.
1906	Robinson, David	Boston.
1906	Rogers, Mark Homer	Boston.
1906	Rowe, Carleton Allen	Tewksbury.
1906	Rushford, Edward Allan	Salem.
1905	Russell, Charles Bradley	Boston.
1905	Russell, Walter Burton	Springfield.
1906	Ryder, Delano Richmond	Fall River.
1906	Scannell, James Joseph	Boston.
1906	Schmidt, Richard Diedrich	Dorchester.
1905	Schofield, Otho Lester	Wellesley.
1905	Seaver, Edwin Pliny, Jr.	Waban.
1905	Sedgewick, Otis White	Wales.
1906	Shaw, Henry	Boston.
1906	Shea, Michael Ignatius	Chicopee Falls.
1906	Shohan, Joseph	Roxbury.
1905	Shoninger, Lee Simon	Boston.
1905	Sleeper, Frank Warren	Dorchester.
1906	Smith, Stafford Baker	Haverhill.
1905	Smyth, Patrick Somers	Boston.
1905	Staples, Clarence Hathorne	Malden.
1906	Stevens, Harold Elmer Ellsworth	Boston.
1905	Stickney, Whitman Gibson	Beverly.
1905	Stone, Ralph Edgarton	Beverly.
1905	Storey, Thomas Andrew	Boston.
1906	Storrs, Henry Randolph	Boston.
1905	Streeter, John Frank	Springfield.
1905	Sturnick, Max	Roxbury.
1906	Sullivan, Cornelius Augustine	South Braintree.
1905	Sullivan, Joseph Lawrence	Roxbury.
1905	Sylvester, Albie Warren	Pittsfield.
1906	Talbot, Fritz Bradley	Brookline.
1906	Thompson, George Hocken	North Adams.
1906	Tinkham, Oliver Goldsmith	Weymouth.
1905	Tobey, George Loring, Jr.	Boston.
1906	Tolman, Henry, Jr.	Boston.
1906	Toohey, Thomas Victor	Roxbury.
1905	Tower, Freeman Augustus	Worcester.
1905	Tracy, John Matthew	Springfield.
1906	Trainor, Joseph Aloysius	Cambridge.
1906	Trask, John Wilson	Lynn.
1906	Trayes, William Henry, Jr.	Boston.
1905	Turner, Charles Humphrey	Haverhill.
1906	Turner, William George	Fall River.
1905	Tyler, Winsor Marrett	New York, N. Y.
1906	Tyzzer, Ernest Edward	Wakefield.
1905	Walker, William Dacre	Boston.

1905	Warren, Edward Dane	Holyoke.
1905	Warren, Franklin Lafayette	Bridgewater.
1906	Way-Allen, Eleanor	Walpole.
1906	Whelan, Charles	Boston.
1905	Whitcher, Burr Royce	Boston.
1906	Whitman, Luther Oakes	Melrose Highlands
1905	Whitney, Ray Lester	Ashburnham.
1906	Whittemore, Wyman	Boston.
1906	Wilinsky, Charles Francis	Boston.
1906	Wilkinson, William Macdonald	Lawrence.
1906	Wilson, John Edward	Boston.
1905	Young, Ralph Randal	Roxbury.
1905	Young, Roy Demas	Arlington.
1906	Zimmermann, Louisa Juliet	Worcester.

List of Deceased Fellows.

Admitted.	Name.	Residence.	Date of Death.	Age.
1885	Barnard, Rebecca	Worcester	Sep. 9, 1905	68
1867	Bass, William	Dedham	July 21, 1905	95
1898	Beattie, Robert Fowler	Brookline	Aug. 6, 1906	32
1893	Blake, Warren Perkins	Springfield	Feb. 15, 1906	47
1843	Brooks, Samuel Doolittle	Springfield	Feb. 26, 1906	89
1893	Burrell, Benjamin Henry	Roxbury	April 23, 1906	53
1871	Chadwick, James Read	Boston	Sep. 24, 1905	60
1874	Chase, Eli Ayer	Brockton	Oct. 3, 1905	58
1861	Curtis, Hall	Boston	June 1, 1906	71
1870	Davis, Wesley	Worcester	Feb. 16, 1906	64
1895	Dever, Charles Edward	Lynn	Oct. 24, 1905	43
1846	Doane, George Washington	Hyannis	Dec. 28, 1905	81
1896	French, John Innes	Winchester	Nov. 18, 1905	44
1882	Galligan, Edward Francis	Taunton	Sep. 26, 1905	48
1866	Gardner, William Wallace	Springfield	Dec. 11, 1905	79
1880	Garrigan, Thomas James	No. Brookfield	April 20, 1906	51
1874	Gilfillan, Thomas	Northampton	Sep. 9, 1905	77
1878	Harriman, James Lang	Hudson	Dec. 28, 1905	78
1895	Hedenberg, James	Medford	July 25, 1905	74
1896	Holland, James William	Westfield	Dec. 29, 1905	37
1856	Hyde, George Smith	Boston	Dec. 11, 1905	74
1875	Kingsbury, Joseph Byron	Holbrook	March 24, 1906	71
1898	Lockwood, George Bertrand	Sharon	July 12, 1905	31
1897	Mahon, James Arthur	Dorchester	Sep. 14, 1905	40
1880	Mellen, William Michael Edward	Chicopee	May 14, 1906	58
1891	Moulton, Charles Fred	West Roxbury	April 24, 1906	40
1860	Munsell, George Nelson	Harwich	Nov. 1, 1905	69
1867	Park, John Gray	Groton	Aug. 29, 1905	67
1893	Parker, Charles Edwin	Princeton	July 10, 1905	41
1897	Pegram, John Combe, Jr.	Providence, R.I.	April 26, 1906	35
1899	Pettee, John Harris	Roxbury	March 5, 1906	34
1875	Pierce, Andrew Martin	New Bedford	Nov. 6, 1905	53
1897	Prefontaine, Louis Aurele	Springfield	June 19, 1905	39
1895	Scannell, Michael Edward	Worcester	Nov. 16, 1905	41

1904	Shead, Edward Wadsworth.....	Cambridge.....	Aug.	19, 1905	31
1895	Sheahan, Joseph Maurice.....	Quincy.....	Sep.	21, 1905	53
1895	Van Rensselaer, Henry Rensselaer	Lanesborough..	Feb.	13, 1905	51
1863	Warner, Emerson.....	Worcester.. ...	Dec.	24, 1905	74
1849	Williams, Jacob Lafayette.....	Boston	May	16, 1906	82
1871	Wood, Edward Stickney.....	Boston.....	July	11, 1905	59
1904	Zabriskie, Fred Templeton.....	Pittsfield.....	Nov.	5, 1905	33

The following names have been changed:

Lootz, Emma to Erving, Emma.
Patch, Anne S. K. to Patch-Lindquist, Anne S. K.

The following has been restored to the privilege of membership in the Society:

Smith, Thomas Delap . . Roxbury.

The following have been placed on the retired list:

Cornelius A. Ahearne, of Lynn.
Chester M. Barton, of Hatfield.
Seabury W. Bowen, of Fall River.
George A. Bragdon, of Middletown, Conn.
John A. Douglass, of Amesbury.
Isaac H. Hazelton, of Wellesley Hills.
Francis A. Howe, of Newburyport.
William W. Leavitt, of Pittsfield.
Nomus Paige, of Taunton.
George E. Pinkham, of Lowell.
N. Morton Ransom, of Somerville.
Albert R. Rice, of Springfield.
Isaac W. Starbird, of Roxbury.
George S. Stebbins, of Springfield.

The resignations of the following have been accepted:

John B. Beebe, of Great Barrington.
Annie M. Blossom, of North Adams.
Zelly A. Bonoff, of New Haven, Conn.
Mary J. Brewster, of Northampton.
Charles Burleigh, of Malden.
M. Augusta Camp, of Northampton.
Elliott M. Clarke, of Denver, Colo.
Henry A. Cooke, of Providence, R. I.
Margaret E. V. Fraser, of Denver, Colo.
Arthur H. Harrington, of New York, N. Y.
Claude P. Jones, of Vineland, N. J.
John G. W. Knowlton, of Exeter, N. H.
Charles S. Little, of Laconia, N. H.

Herman P. Marshall, of Deming, Wash.
Homer T. Partree, of Eatontown, N. J.
Jay Perkins, of Providence, R. I.
Charles G. Stearns, of East Dennis.
Alexander T. Walker, of West Somerville.
Frederick B. Willard, of Hartford, Conn.
Louis F. Wheatley, of Meriden, Conn.

The following have been deprived of the privileges of membership for non-payment of dues:

Bradford Allen, of Nashua, N. H.
Edwin W. Bullock, of Haverhill.
Theodore C. Burnett, of San Francisco, Cal.
Frank F. Carr.
Mary V. Church, of Topeka, Kansas.
Joseph E. Clark, of New York, N. Y.
Charles F. Cogswell, of Brookline.
Joseph M. Conlin, of Chicopee.
Samuel P. Cottrell, of Newport, R. I.
Francis X. Crawford, of Boston.
Thomas J. B. Dillon, of Roxbury.
Benedict Donovan, of Brockton.
Edward A. Drummond, of Providence, R. I.
Whitfield O. Dunham, of Sioux Falls, S. D.
George F. Garland, of Waltham.
George W. Grover, of Sheffield.
Ira C. Guptill, of Northborough.
Revere R. Gurley, of New York, N. Y.
Rufus H. Hall, of Everett.
Charles C. Holcombe, of Lee.
Dixi G. Hoyt, of Providence, R. I.
Anselme E. Houle, of Bennington, Vt.
Rufus P. Hubbard.
Charles Storer Knight, of Woodfords, Me.
Edwin Leonard, Jr., of Jersey City, N. J.
George C. Littlefield, of Salem.
Ernest R. Mackintosh, of Lowell.
Jared H. Miller, of Palmer.
Philip W. T. Moxom, of Flatbush, N. Y.
Charles O. Murphy, of New York, N. Y.
George M. Muttart, of East Boston.
Frank H. Parker, of Malden.
Luke B. Peck, U. S. Army.
Harry M. Peggs, of Somerville.
Winfred C. Pike, of Dorchester.
Harris S. Pomeroy, of Peabody.

Eustace P. Reid.
Edwin W. Rich, U. S. Army.
Charles C. Rothfuchs, of Boston.
Oliver M. Sheridan, of Jamaica Plain.
Frank E. Smart, of Lowell.
Warren B. Stone.
Harry G. Wyer, of Kirkwood, Mo.

The following has been expelled:

Percy DeMille McLeod, of Boston.

SUPPLEMENT No. III. TO CATALOGUE OF 1905.

Admissions reported from June 12, 1906, to June 11, 1907.

1906	Adams, James Thacher	Boston.
1906	Bain, John Baxter	Lawrence.
1906	Bannon, John Hugh	Lawrence.
1907	Barrett, Edward William	Medford.
1906	Barron, Elmer Walter	Charlestown.
1906	Barstow, Andrew Thaddeus	Fitchburg.
1907	Bates, Lewis Beals	Boston.
1906	Beauchamp, Joseph Octave	Chicopee Falls.
1906	Beaulieu, Francis Xavier	Taunton.
1907	Beeley, Leon Gage	Lawrence.
1906	Bellehumeur, David Stanislaus	Lowell.
1907	Berwick, James Roderick	Methuen.
1907	Bessey, Earle Emerson	Hathorne.
1907	Bier, Max Daniel	Lawrence.
1907	Boothby, Walter Meredith	Boston.
1906	Boyle, Jeremiah Joseph	Cambridge.
1907	Brainerd, Walter Scott	Bradford.
1907	Bruce, Harold Milton	Brookline.
1907	Bullard, Edward Arthur	Cambridge.
1906	Burgess, Charles James	Lawrence.
1906	Bushold, Fred George	Lawrence.
1906	Campbell, Matthew Philip	Provincetown.
1906	Carley, Frederic James	New Bedford.
1906	Chalmers, Hattie Elizabeth	Boston.
1907	Champion, Merrill Edwin	Arlington.
1906	Chapin, Laurence Dudley	Springfield.
1906	Clancy, Daniel Francis	Hopkinton.
1907	Clark, William Irving, Jr.	Worcester.
1907	Cobb, Chester Thompson	Easthampton.
1906	Conlon, Frank Aloysius	Lawrence.

1907	Connor, William Henry	Springfield.
1906	Conroy, Edward Cornelius	Andover.
1907	Coon, William Hall	Lawrence.
1906	Corbett, Jeremiah Joseph	Boston.
1907	Coriat, Isidor Henry	Boston.
1906	Cornwall, Anna Marlin	Boston.
1907	Cotter, Edward Joseph	Boston.
1907	Cowan, Marion	Lynn.
1907	Cox, Ann Caroline	Jamaica Plain.
1907	Crispo, Peter Timothy	Fall River.
1906	Cusick, Lawrence Francis	Boston.
1906	Cyr, Emile Edouard	Lawrence.
1907	Dascombe, Otho Lee	Waltham.
1906	Davis, Ernest Leland	Springfield.
1906	Day, Cushman	Boston.
1907	Deacon, Thomas Irving	Cambridge.
1906	Deems, Owen Manfred	Springfield.
1906	Dewing, Louis Alexander	Boston.
1907	Dickinson, Gyrdie Willie	Jamaica Plain.
1907	Dowling, Charles Joseph	Springfield.
1906	Downing, Andrew Francis	Watertown.
1906	Dutra, Emmanuel Christian	Taunton.
1906	Eaton, William Edward	Roxbury.
1906	Felch, Carrie Innes	Boston.
1906	Felch, Lewis Perley	Boston.
1907	Finegan, Daniel Joseph	Gloucester.
1906	Frothingham, Channing, Jr.	Boston.
1907	Fuller, Ernest Page	Lawrence.
1907	Gahan, Patrick Francis	Medford.
1906	Gallagher, John Henry Connaughton	Chicopee.
1907	Gallagher, Thomas Morton	Newton.
1906	Galligan, Edward Joseph	Taunton.
1907	George, Arial Wellington	Boston.
1907	Giblin, Francis Joseph	Dorchester.
1906	Giroux, Charles	Three Rivers.
1906	Goldsbury, Paul Williams	Boston.
1907	Goldthwaite, Ralph Harvard	Worcester.
1906	Goodell, William	Springfield.
1906	Green, Robert Montraville	Boston.
1906	Hager, William Perry	Northampton.
1907	Hamilton, Frank Andrew	Somerville.
1906	Hanson, William Thomas	Worcester.
1907	Hatch, Ralph Augustus	Brookline.
1906	Heffner, William James	Northampton.
1906	Henry, Thomas Francis	Worcester.
1907	Hoit, Henry Ambrose	Holden.
1906	Holmes, George Winslow	Boston.

1907	Holt, Charles Herbert	Salem.
1906	Houghton, Richard Henry	East Boston.
1907	Hoyt, Charles Wentworth	Boston.
1906	Hughes, Patrick Joseph	Lawrence.
1907	Jones, Everett	Brookline.
1906	Joyce, Thomas Francis	Lawrence.
1907	Kazanjian, Hampar Paul	Lawrence.
1907	Kent, Bradford	Dorchester.
1907	Kirkpatrick, George Holland	Lynn.
1906	Landers, George Bagnell	Chelsea.
1907	Lawlor, Edward Francis	Lawrence.
1906	Lawlor, Richard Henry	Methuen.
1907	Laws, Sophie Goudge	South Boston.
1906	Leary, Timothy	Roxbury.
1906	Lemieux, Theodule Alfred	Lawrence.
1907	Levy, Felix Julius	Roxbury.
1906	Lloyd, Henry Demarest	Boston.
1906	Lowney, Dennis Joseph	New Bedford.
1907	Lyman, William Robinson	Worthington.
1906	McArdle, John Joseph	Lawrence.
1906	McAvoy, John Joseph	Lawrence.
1907	MacCallum, Wallace Peter	Dorchester.
1907	McCarthy, Louis Flurence	Malden.
1906	McCurdy, Theodore Edward Alexis	Roxbury.
1906	MacKenzie, Lewis Francis	Wellfleet.
1907	Mackinnon, Donald Lauchlin	Boston.
1907	Maloney, Daniel Arthur	Watertown.
1906	Maney, John Joseph	Lawrence.
1907	Masse, John Baptiste	Lawrence.
1907	Mathes, Roy Wentworth	Lynn.
1906	Mehan, Joseph Aloysius	Lowell.
1907	Meyer, Carl Emil	Springfield.
1907	Mignault, Louis Joseph Adolphe	Fall River.
1907	Monahan, John Terence	Fall River.
1907	Morrison, Archibald Benjamin	Boston.
1906	Mullin, Norbert Vincent	Boston.
1907	Myers, Elizabeth Young	Roxbury.
1907	Nevers, Harry Hill	Lawrence.
1907	Norton, Daniel Capron	Salem.
1906	O'Brien, William Smith	Chicopee.
1907	O'Connor, Thomas Hugh	Roxbury.
1907	O'Reilly, James Archer	South Boston
1907	Packard, Horace	Boston.
1907	Paddock, Brace Whitman	Pittsfield.
1907	Peirce, Bradford Hendrick	Cambridge.
1906	Penhallow, Dunlap Pearce	Boston.
1907	Perkins, Thomas Tounge	Cliftondale.

1906	Petit, Alphonse Herbert	Lawrence.
1907	Phelps, Olney Draper	Worcester.
1907	Phillips, Wilson Frank	Dorchester.
1907	Place, Edwin Hemphill	Boston.
1906	Plunkett, Harold Brabazon	Lowell.
1906	Porter, Fred Knight	Southwick.
1907	Potter, John Claude	South Framingham.
1907	Raymond, Charles Stanley	Northampton.
1907	Redmond, Thomas Henry	Lawrence.
1907	Reid, Isadore Eugene Rosenstein	Jamaica Plain.
1906	Richardson, Horace Kimball	Bradford, Me.
1906	Riordan, Walter Daniel	Lawrence.
1906	Risley, Edward Hammond	Boston.
1901	Rockwell, Alfred Elijah Perkins	Worcester.
1907	Rood, Luther Colby	Dorchester.
1907	Roughan, Charles Michael	Lowell.
1906	Roy, Joseph Napoleon	Webster.
1906	Russell, Frederick James	Newton Centre.
1907	Sawyer, Alpha Reuben	Roslindale.
1907	Schwartz, Myer	Lawrence.
1906	Shanahan, Edward Joseph	Taunton.
1907	Shaw, Adam Ernest	Lowell.
1907	Sherman, Warren Hunter	Westford.
1906	Slutskin, Maurice Louis	Springfield.
1906	Smith, John Hall	Boston.
1906	Smith, Myrtle	Worcester.
1907	Southgate, Alfred Willard	Worcester.
1907	Sparrow, Ernest Harold	Cambridge.
1907	Stammers, John Collins	Charlestown.
1907	Stockbridge, Alberto Horatio	Lynn.
1907	Sullivan, Charles Brent	Hathorne.
1906	Sullivan, John Joseph	Lawrence.
1906	Thayer, Nathaniel Pulsifer	Boston.
1907	Thurber, Madison Templeton	Dorchester.
1906	Tucker, Arthur Wallace	Worcester.
1907	Turner, Charles Sampson	Boston.
1906	Walch, Joseph Francis	Lawrence.
1906	Walcott, William Wright	Natick.
1907	Wardwell, James Knight	Roxbury.
1907	Watters, William Henry	Boston.
1907	Watts, Joseph Palmer	Salem.
1907	Wells, David Washburn	Boston.
1906	Wells, Orion Vassar	Westford.
1906	Wilder, Winford Oliver	Pittsfield.
1906	Williams, John Thomas	Boston.
1906	Young, Evangeline Wilson	Boston.
1907	Young, James Herbert	Roxbury.

List of Deceased Fellows.

Admitted.	Name.	Residence.	Date of Death.	Age.
1852	Barrett, William Marshall	Westborough	Nov. 4, 1906	84
1839	Bemis, Charles Vose	Medford	Nov. 6, 1906	90
1874	Bowker, Charles	Bernardston	Dec. 10, 1906	82
1899	Brown, Alphonso Bickford	Newburyport	Oct. 17, 1906	34
1867	Butler, Winthrop	Vineyard Haven	April 23, 1907	68
1900	Carothers, William Harsha	Melrose	Oct. 23, 1906	51
1847	Davis, Robert Thompson	Fall River	Oct. 29, 1906	83
1871	Dearborn, Alvah Burton	Somerville	Aug. 19, 1906	64
1866	Dudley, Henry Watson	Abington	Dec. 29, 1906	75
1862	Everett, James Bradley	Everett	May 20, 1907	78
1875	Finn, James Anthony	Roxbury	Sept. 1, 1906	66
1870	Gaffney, Henry Joseph	Salem	Feb. 9, 1907	59
1881	Hall, Walter Langdon	Medford	Oct. 25, 1906	60
1861	Harlow, John Martyn	Woburn	May 18, 1907	87
1850	Heard, John Theodore	Boston	Sept. 2, 1906	70
1844	Hubbard, Benjamin	Plymouth	Jan. 12, 1907	89
1899	Lecompte, Walter Augustus	Boston	Jan. 13, 1907	36
1892	Lyons, Christopher Philip	Boston	Jan. 22 1907	37
1864	McDougall, Samuel Jay	Boston	Feb. 8, 1907	76
1882	Mackie, George	Attleborough	Aug. 31, 1906	56
1891	McQueeny, Francis Joseph	Boston	Oct. 1, 1906	48
1859	Mansfield, John Robbins	Wakefield	Nov. 11, 1906	74
1841	Mansfield, Joseph D.	Wakefield	May 22, 1907	90
1876	Marion, Otis Humphrey	Allston	Nov. 27, 1906	59
1901	Odiorne, Walter Burlingame	Boston	July 14, 1906	33
1871	Odlin, Charles Cushing	Melrose	Dec. 18, 1906	59
1869	Paun, Amos Bosworth	Middleborough	April 6, 1907	60
1881	Pitcher, Samuel	Hyannis	Feb. 23, 1907	82
1860	Provan, Robert	Brookline	Feb. 23, 1907	73
1906	Roberts, Frederick Albert	Wilmington	Aug. 11, 1906	33
1877	Seymour, James Dwight	Whately	Sept. 10, 1906	56
1884	Spring, Clarence Walter	Fitchburg	Nov. 2, 1906	47
1886	Starbird, Isaac Warren	Roxbury	Feb. 2, 1907	67
1866	Stickney, Alonzo Lawrence	Ashburnham	June 4, 1907	72
1882	Tanner, John Alexander	Dorchester	June 21, 1906	53
1879	Watson, George Henry	Brooklyn	May 25, 1907	59
1896	White, Leonard Darling	Uxbridge	Sept. 18, 1906	47

The following has been elected an honorary member:

1907 MacMonagle, Beverly . . San Francisco, Cal.

The following names have been changed:

Austin, Mabel Fletcher, to Southard, Mabel Austin.
Lilienthal, Alice, to Hirshberg, Alice Lilienthal.

The following have been restored to the privileges of membership:

Bradford Allen, of Nashua, N. H.
Francis Xavier Crawford, of Boston.
Edwin Putnam Gleason, of Brockton.
Winfred Carle Pike, of Boston.

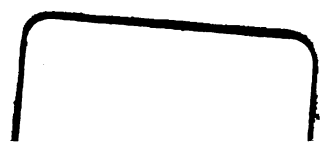
The following have been placed on the retired list:

Robert Amory, of Boston.
Charles B. Bridgham, of Cohasset.
John P. Brown, of Taunton.
David W. Cheever, of Boston.
Edward R. Cogswell, of Cambridge.
Samuel A. Davis, of Somerville.
John G. Dearborn, of Charlestown.
Robert Disbrow, of Boston.
William W. Eaton, of Danvers.
Hugh Ferguson, of South Boston.
Linnæus V. Gibbs, of Huntington.
Eugene A. Gilman, of Roxbury.
Thomas Hall, of Boston.
Horace G. Leslie, of Amesbury.
Horace P. Makechnie, of West Somerville.
William M. Mercer, of Pittsfield.
Albert L. Norris, of Cambridge.
Hamilton Osgood, of Boston.
Charles B. Porter, of Boston.
Octavius B. Shreve, of Salem.
George E. Stackpole, of Malden.
Lincoln R. Stone, of Newton.

The resignations of the following have been accepted:

Melvin P. Burnham, of Ray Brook, N. Y.
Donald Churchill, of Providence, R. I.
Francis J. Dore, of Roxbury.
Charles W. Eveleth, of New York, N. Y.
James T. Fisher, of Los Angeles, Cal.
Sherwin Gibbons of Los Angeles, Cal.
George H. Gleason, of Dorchester.
James F. Hall, U. S. Army.
Benjamin J. Handy, of Fall River.
Robert B. Harrington, of Northampton.
Edward S. Hatch, of New Orleans, La.
William E. MacCoy, of Brookline.
Beverly MacMonagle, of San Francisco, Cal.
William R. May, of New York, N. Y.
George W. Nash, of Hurley, N. Y.
Harry C. Parker, of Indianapolis, Ind.
Russell S. Rowland of Detroit, Mich.
Otis W. Sedgewick, of Brookfield, Conn.
Alice W. Tallant, of Philadelphia, Pa.
Elmer S. Tenney, U. S. Army.

The following have been deprived of the privileges of membership for non-payment of dues:

Charles E. Bedell, of Somerville.
Joseph T. L. Brennan, of Lowell.
Daniel R. Brown, of Salem.
Thomas F. Carroll, of Boston.
William E. Carroll, of Taunton.
Robert R. Culbert, of Roxbury.
Charles H. Curran, of Holyoke.
Frederick A. Davis, of Boston.
Charles F. Denny, of St. Paul, Minn.
John A. Elmere, of Los Angeles, Cal.
Emma L. Erving, of Washington, D. C.
William G. Erving, of Washington, D. C.
Ammen Farenholt, U. S. Navy.
Nathan L. Griffin, of Bradford, N. H.
Newbert J. Hall, of Brookline.
Alvin D. Holmes, of Hyde Park.
George P. Hurd, of Holbrook.
Thomas L. Jenkins, of Topsfield.
Harold J. Levis, of Rochester, N. Y.
Dwight M. Lewis, of New Haven, Conn.
George F. Lewis, of New Bedford.
James R. Matheson, of Roxbury.
Laura T. Myers, of Allston.
Philippe H. Nichol, of Worcester.
Clarence C. Northrop, of Manchester, N. H.
Payn B. Parsons, of New York, N. Y.
Archibald J. Ranney, of Cleveland, O.
Wilbur B. Robertson, of Worcester.
Frank R. Searles (residence unknown).
Hiram H. Seelye, of Daytona, Fla.
George A. Spencer, of Haverhill.
Maurice J. Treston (residence unknown).
Kenelm Winslow, of Seattle, Wash.

3 gal
224+